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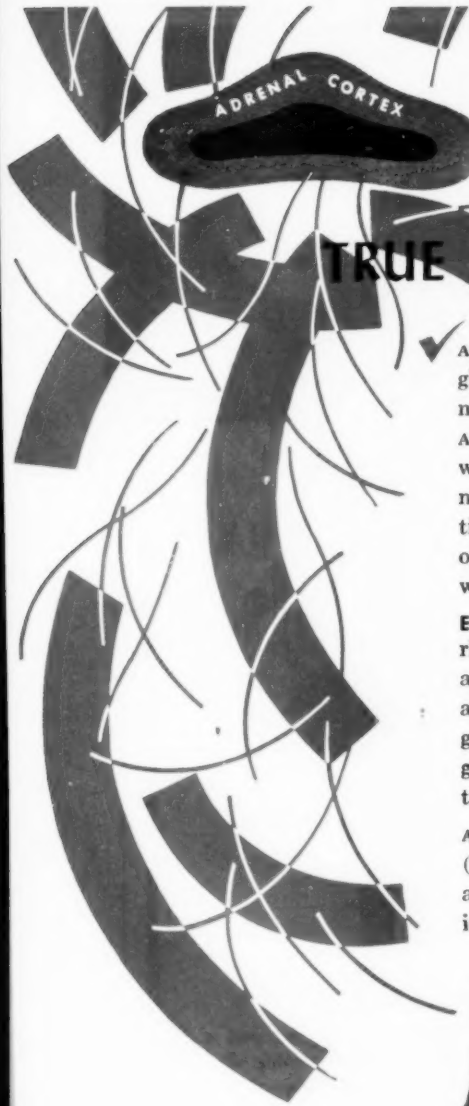

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South African Medical Journal
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HEPATITIS AND THE POST-HEPATITIS SYNDROME*

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Patients who have recently suffered from hepatitis show a series of symptoms, which are summarized in medical literature as the 'Post-Hepatitis Syndrome'. They were first described in 1944 by Caravati, afterwards by Benjamin and Hayt. In 1946 Sheila Sherlock concluded that the symptoms were not related to disturbed liver functions but that the condition was a neurosis founded on a psychogenic basis. During and after the war the number of cases of infectious hepatitis increased considerably and our knowledge about their origin has changed considerably too. From most parts of the world (and especially from those countries involved in the war) reports arrived concerning a considerable increase of this parenchymatous affection of the liver. The old conception of catarrhal icterus has undergone a considerable extension. Already for more than half a century it has been accepted, mostly on the authority of Virchow, that catarrhal icterus was caused by an obstruction of the ductus choledochus through swelling of the mucous membrane. Nowadays we speak of infectious hepatitis or infectious icterus and we know that it is not a disease of the outlets of the gall bladder but an affection of the liver parenchyma.

It is understandable that formerly, when the catarrhal icterus was ascribed to a swelling of the mucous membrane of the duodenum and the infectious icterus to a hepatitis, both diseases were considered as quite different conditions. It is equally understandable that at present (since it is certain that both depend upon a hepatitis) many investigators consider the so-called catarrhal icterus and the infectious icterus (the symptoms of which show such a large resemblance) as an infectious disease, caused by one and the same micro-organism. At present it is generally accepted that in these cases one is faced with a filtrable virus, which is already present in the blood before the clinical symptoms show themselves and that it remains there, long after the jaundice has disappeared.

As a rule, nowadays, two forms are distinguished:

1. *Serum Jaundice.* This develops about 60-150 days after the patient has been injected with human serum, in which the agent causing the disease was present.

Extremely slight traces are sufficient, so that not only blood transfusions and serum injections, but also insufficiently disinfected syringes used for venepuncture or vaccination scarifiers and the needle of Fränkel, used for pricking the skin in order to obtain blood samples, can transmit the virus. In these patients the virus would not be present in stool or urine, so that it would not result in contact infections.

A good idea of the frequency of transmission of this virus through the hands of the medical man may be obtained from the painstaking investigation in the Scandinavian countries. The patients admitted as suffering from this disease were split into two groups. The first group comprised those who, during the previous six months, had not been admitted to a hospital or had not been injected at home on account of some disease or other, or had not been vaccinated. The other group had been in contact with the needle of the doctor. This investigation showed that the latter group was 40 times larger than the first one and that the larger number of cases of infectious icterus had been transmitted by medical intervention. Without doubt this is a disturbing fact, especially when one considers that this disease is far from innocent and not only may result in degeneration of the liver or liver atrophy, but also is the cause of many cases of cirrhosis, which only much later show clinical symptoms. In recent years we have also seen many cases of hepatitis where transmission of the virus has occurred by medical hands.

The long period, at least 2 and often 4 to 5 months, between the introduction of the virus and the time when the disease manifests itself, is probably the reason why it took such a long time before this connexion became clear to us. But certainly it is a serious warning to medical men that everything used to inject a patient or draw blood from him, may only take place with thoroughly sterilized instruments. Keeping syringes and needles in alcohol has been shown to be not enough. Everything we use for this purpose needs adequate boiling.

2. *Infectious Jaundice.* Here there is contact with other patients via stool and urine. This form was observed particularly in camps in Germany and Indonesia. The incubation period is from 18-40 days. Here, also, a filtrable virus is the cause. This virus is present in the

* Report of a lecture delivered at a meeting sponsored by the Cape Town Post-Graduate Medical Association, the Cape Western Branch of the Medical Association of South Africa and the University of Cape Town, on Tuesday, 15 May 1951.

blood in the pre-icteric stage and appears in the stool in the active stage and during convalescence. The infection usually spreads from here. The nasopharynx and respiration appear not to be of importance for transmission. Whether immunity ensues is not certain. Many persons, infected simultaneously with the virus, become only slightly ill and a visible icterus is not invariable. Chemically an increase of the bilirubin in the serum is demonstrable.

The question is now whether one has to deal with a single virus or with different kinds of filtrable viruses. Most observers are inclined to the opinion that there are different kinds. Yet one asks oneself from which source the patient became infected, when his blood afterwards appears to be able to cause in another a disease so exactly similar. It is possible, one must say, that the characteristics of the virus change according to whether it has undergone a phase in the stool or has stayed for long in the blood of the host.

It is said that the clinical aspects cannot be distinguished from one another. Yet I would like to draw your attention to this point. In the old picture of the disease, the catarrhal icterus, we have learned that it started with gastro-intestinal symptoms, with dyspepsia, sometimes vomiting; in short, symptoms of the stomach and the upper part of the small intestine. When icterus appeared, in most cases there was a short period of discoloured, putty-like faeces, lasting only 1-3 days. It was these symptoms which caused the opinion that the mucous membrane of the swollen duodenum obstructed the ductus choleduchus. Now that we know that we are faced with a hepatitis, we often do not mention these symptoms, but whether we have to deal with a hepatitis or not, the fact remains that there is a temporary obstruction of the biliary passages. There is also reason to suppose that in these cases the oral infection first caused a gastro-duodenitis and that subsequently an affection of the liver parenchyma occurred, probably via the vena porta to the liver. In any case, from the clinical picture one does not get the impression of an increasing cholangitis.

In the case of serum hepatitis this feature of the disease, as far as I have been able to control my patients, is lacking. Here the icterus sometimes appears abruptly, sometimes more slowly; fever appears and the patients feel ill. Perhaps in a single case there is some nausea, as happens in many cases of other infectious diseases, but not the typical gastro-intestinal symptoms of catarrhal icterus and certainly no period of acholic stools. The infectious form always shows these gastro-intestinal symptoms and in a high percentage of cases a more or less obvious period of acholic stools.

I will not deal with the clinical symptoms themselves or with the complications which appear after the disease. It is with the post-hepatitis syndrome that I wish to deal:—

1. I shall commence with a discussion of a few patients. The first patient is a woman of 44 years. Eight months ago she had a serum hepatitis. That it had been a serum hepatitis we may be allowed to deduce from the absence of the gastro-intestinal symptoms and the absence of her acholic phase. Moreover, she had been in the hospital three months before that for a prolapse operation. After the operation she had a blood transfusion and many injections. Until the operation she had always felt well and she was quite capable of attending to her busy household affairs. The fever subsided rapidly and the jaundice disappeared after 2 or 3 weeks but she never felt completely well again. The main complaint was one of being immensely tired. This feeling of fatigue not only appeared after she had been busy, but also when she had been sedentary. Apart from that she felt listless and she also tired quickly mentally. When she read a book, she soon put it aside, especially when the reading matter was somewhat difficult. She had become nervous and irritable. She felt chilly, when before she did not, and always wanted a hot water bottle in bed in the evening, because otherwise she could not get warm and could not fall asleep. She perspired easily, but not over the entire body, mainly on her face and hands. Her appetite was moderate, but her weight remained about constant. Her meals were indigestible; often she had a somewhat heavy, swollen feeling in the region of the liver, but no pains.

Since nothing abnormal could be found, and the erythrocyte sedimentation rate was normal, her complaints were considered to be of a psychasthenic nature. This contributed mainly to making her more nervous.

On general examination nothing abnormal was found. The liver was palpable, perhaps still somewhat enlarged, but not painful. The sedimentation rate was normal. Nothing abnormal was found in the lungs. No rise in temperature. Pulse normal. Blood pressure 110/80 mm. Hg. There was a little urobilin in the urine. The bilirubin content of the serum was normal. Various liver function tests showed no abnormal results. Cholesterol, 165 mg. per 100 c.c. with an esterification of 50 mg. (±30%). The blood sugar curve was strikingly flat. The stomach acid values were low and the mucosa reacted only slowly to histamine. The basal metabolism in the closed system was −21%.

2. The second patient was a young man of 28 years. Six months ago he suffered from hepatitis. Before that he had not been under medical treatment. This disorder started with gastro-intestinal symptoms and acholic faeces; probably, therefore, a hepatitis caused by direct infection. The icterus lasted about 4 weeks. Since then the patient remained tired and weak. Now and then the liver region was somewhat tender, but the feeling of fatigue and of listlessness were the worst symptoms. He also complained of feeling the cold, became nervous, but not irritable. His appetite was moderate, otherwise he had no complaints. The liver was still somewhat enlarged, but not painful. Blood pressure 120/85 mm. Hg. B.M.R. −16%. Cholesterol esterification 42%. Flat blood sugar curve. Diminished hydrochloric acid values in the stomach. Serum bilirubin normal. Liver function tests practically normal.

These are two typical examples of cases described as the post-hepatitis syndrome. The feeling of great fatigue and listlessness always constitutes the main complaint. The other complaints are nearly all of a secondary nature. We have seen many of these patients in Germany and in Indonesian camps, but also amongst the free people of the Netherlands. It is a syndrome which is quite prevalent and sometimes continues for a long period, even years after the hepatitis has occurred. In other cases it passes quickly, but almost every patient, who has had a hepatitis,

has a shorter or longer period of this fatigue, more so than is the case in other infectious diseases. Even patients who suffer from such a slight hepatitis that their icterus can hardly be seen and then only for a short period, sometimes complain about this fatigue. Sometimes it is this tiredness which makes them see the doctor and which enables the latter to discover the earlier hepatitis. Usually the disability subsides quickly and spontaneously, but with other patients it may last months and even years. Whether in the latter case we have to do with precursors of a cirrhosis which may develop later, cannot yet be stated. For this the time of observation has been too short.

Other authors have always confined themselves to doing a few liver function tests. These have usually been found to be normal. Since nothing else abnormal was found, these complaints were often considered as being of a nervous nature or they were given the more general name of post-infectious asthenia, without people trying to realize what the abnormality actually consisted of.

Since in our clinic all patients who suffer from fatigue are investigated in the metabolism chamber, we soon obtained a knowledge of the basal metabolism in cases of the post-hepatitis syndrome. The metabolism is in most cases low, sometimes up to −25%. Even in the case of patients, who had become nervous and irritable and where one would think of a slight hyperthyroidism, as a rule a lowered basal metabolism was found.

Other signs, not constant, are a low percentage of cholesterol esterification. This percentage is nearly always below 50. Comparatively often there is a somewhat flat blood sugar curve. If one measures the blood pressure, one is struck by the fact that the pulse pressure is generally less than normal. The fractional test meal often gives low values, sometimes an achylia, which, however, reacts to histamine.

When the patients recover, and the fatigue disappears, the signs do the same simultaneously and the normal conditions return.

It is somewhat speculative in the course of these more clinical observations, to go deep into the explanation of these signs and symptoms. We only want to mention that the liver plays an important part in fat metabolism, in regard to the combustion of fats, in order to keep the body temperature normal as well as in regard to the esterification of lipoids. But whatever the definite explanation may be, this post-hepatitis syndrome must be considered to be the result of a general disturbance in the centre of which is the function of the liver.

For the moment we will be more concerned with the question: how can we help these people to overcome this difficult period? What is the therapy?

Let me begin by saying that thyroid treatment, instituted in view of the low basal metabolism, in most cases is not very successful. It is true that we have here a hypometabolism, but not a real hypothyroidism. Yet one can try to do it in combination with sympatol or the Netherlands Novosymptom. Increasing doses of thyroid may be given together with this drug and with careful control of the pulse. Furthermore, tonics and such medicaments may be given as well as physical therapy, baths, general irradiation with ultra-violet light. A single careful diathermy application over the liver is also to be recommended. In some cases patients react well to treatment with liver preparations.

As regards diet, we consider that food poor in fat and rich in albumin and carbohydrates is the most suitable. Long periods of rest more often have a negative rather than a positive result. When the icterus has disappeared and when the liver function tests indicate no definite damage to the liver cell, a regular and active life is better than giving in to the feeling of fatigue as a means of achieving something through rest cures.

ABSTRACTS

Radical Cure of Relapsing Vivax Malaria with Pentaquine-Quinine: a Controlled Study. B. Straus and J. Gennis (1950): Ann. Int. Med., **33**, 1413.

Fifty veterans of World War II who were admitted to a Veterans Administration Hospital in New York for relapsing vivax malaria were treated with a daily dose of 30 mg. pentaquine base (= 40 mg. pentaquine phosphate), combined with 1.8 gm. quinine sulphate, for 14 days.

A control series of 49 patients was treated with chloroquine, because this otherwise highly effective drug has no significant effect on the relapse rate.

All patients suffered from vivax malaria since two years, during which period they had had an average of five relapses, prior to admission in the aforementioned hospital.

After treatment had been concluded nearly all patients could be followed-up for at least one year; the longest follow-up period was 18 months.

Only one patient out of the fifty in the pentaquine-quinine series relapsed during the follow-up period (2 per cent), after 115 days. He was then given two courses of mepacrine which failed to eliminate the parasitemia. This might indicate that he was infected with a particularly resistant strain of vivax malaria and this might account for the failure of pentaquine-quinine.

Seventeen patients out of 49 of the chloroquine group relapsed (35 per cent); one patient had four relapses, two patients had two, and 14 patients had one relapse: 22 relapses in 17 patients.

Toxic manifestations to pentaquine-quinine were minor; in one case quinine was discontinued because of cinchonism. (This might have been prevented if only 1 gm. of quinine sulphate had been administered (an adequate dose for the treatment of vivax malaria), instead of 1.8 gm.—*Reviewer.*)

The authors conclude: In the treatment of P. vivax malaria pentaquine administered concurrently with quinine has proved to be a highly effective curative agent.

Malaria and Pregnancy in Saigon. Le Van Hung (1951): Rev. du Paludisme, **83**, 75.

This important paper emanates from the obstetrical department of the Faculty of Medicine, Saigon. As regards the treatment of malaria in the pregnant patient the author comes to the following conclusions:

1. Quinine does not cause abortion and does not induce labour even when administered in a large dose.

2. Quinine, by suppressing the fever, stops the first uterine contractions and thus prevents abortion or premature confinement.

3. Thus, treatment with quinine makes it possible to the malarious woman to continue her pregnancy to the normal term.

4. Treatment should be energetic, as in all cases of acute malaria, the quinine dosage being 1 gm. to 1.5 gm. a day. This initial treatment should be followed by a consolidation treatment of ten days, in order to prevent the recrudescences which tend to be frequent in pregnancy.

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EDITORIAL

FINGER-PRINTING THE BLOOD

The great increase in the number of the different human blood groups discovered has made a big difference to the chances whereby those falsely accused of paternity can be exonerated.

Today a modern and properly equipped laboratory should be able to call upon at least seven different blood group systems which together could exclude some 62% of men wrongfully accused of fathering children.

The blood groups which can be relied upon to achieve this object, when taken together, are the following:—

1. ABO.
2. MNS.
3. Rh.
4. Kell.
5. Lutheran.
6. Secretion.
7. Duffy.

Most, if not all the antisera are available in South Africa for these blood group tests and while several of them are of recent discovery, they are probably reliable for medico-legal purposes.

The Lewis blood groups have not been included in the list recommended because the Lewis antigens differ in infants and in adults. Reservations must also at the present time be entertained about the Duffy blood group system, because the quantitative studies which would be desirable before crucial evidence from such sources could be legally acceptable, still remain to be undertaken.

Race and Sanger[1] have recently indicated the unique way in which the Kell blood groups may, on rare occasions, prove paternity beyond all reasonable doubt, provided the brothers of the accused person had adequate alibis, because 'outside the family of the accused there would not be another such man in ten million.'

The use of blood groups is, of course, not limited to problems of paternity. Maternity has been excluded on occasion; newly born children, accidentally interchanged in maternity homes, have been identified and restored to their parents; the family relationship of stolen children has been determined and identical twins can be distinguished from non-identical twins.

It is likely that South African Courts would accept the evidence from blood group tests provided these have been performed by persons sufficiently expert in this work. At present there is no way in which any citizen can be

1. *Blood Groups in Man*, p. 275, 1950. Oxford: Blackwell Scientific Publications.

VAN DIE REDAKSIE

BLOED-IDENTIFISERING

Die groot toename in getal van menslike bloedgroepe wat ontdek word, het 'n aansienlike verskil veroorsaak in die moontlikhede dat aangeklaagdes weens vaderskap vrygespreek kan word.

Vandag behoort 'n moderne en goed toegeruste laboratorium by magte te wees om gebruik te maak van ten minste sewe verskillende bloedgroepsisteme, wat gesamentlik sowat 62% van mans kan uitskakel, waar hul per abuis daarvan beskuldig word dat hul die vaders is van kinders.

Die bloedgroepe waarop vir hierdie doel gesteun kan word as hul saam geneem word, is die volgende:—

1. ABO.
2. MNS.
3. Rh.
4. Kell.
5. Lutheran.
6. Secretion.
7. Duffy.

Die meeste, indien nie almal nie, van hierdie antisera is verkrygbaar in Suid-Afrika vir aanwending by hierdie bloedgroeptoetse en hoewel etlikes daarvan maar onlangs ontdek is, tog is hul waarskynlik betroubaar vir geregtelik-geneeskundige doel.

Die Lewis-bloedgroep is nie ingesluit by die lys wat aanbeveel is nie, omdat daar 'n verskil is tussen die Lewis-antigenes van suiglinge en dié van volwassenes. Voorbehoude moet ook voorlopig gemaak word t.o.v. die Duffy-bloedgroepsisteem, want die kwantitatiewe bestudering wat gewens sou wees, vóórdat deurslaggewende bewyse uit sulke bronne vir die gereg aanneemlik kan wees, moet nog aangepak word.

Race en Sanger[1] het onlangs die besondere wyse aangedui, waarop die Kell-bloedgroepe, in seldsame gevalle, die vaderskap bo alle twyfel mag bewys, op dié voorwaarde, dat die broers van die beskuldigde persoon voldoende alibis het, omdat ‚buite die familie van die beskuldigde om daar nie nog so 'n persoon uit tien miljoen sal bestaan nie'.

Die aanwending van bloedgroepe is natuurlik nie net tot vraagstukke oor vaderskap beperk nie. Moederskap is al by geleentheid uitgeskakel daardeur; pasgebore kinders is al uitgeken en aan hul ouers terugbesorg waar hul per abuis in kraaminrigtings omgeruil is; die familieverband van ontvoerde kinders is al vasgestel geword en eenderse tweelinge kan daardeur onderskei word van ander nie-eenderse.

Dit is waarskynlik dat Suid-Afrikaanse geregshowe die getuienis voortspruitende uit bloedgroeptoetse sal aanneem op voorwaarde dat hul uitgevoer is deur persone

1. *Blood Groups in Man*, p. 275, 1950. Oxford: Blackwell Scientific Publications.

compelled by the Court to submit to this kind of examination, but an interesting departure is contemplated, in respect of criminal matters, in an amendment published in the *Criminal Procedure and Evidence Amendment Bill, 1950*. If the provisions of this Bill become law, any person arrested upon any charge may be required to submit to the taking of a blood sample, to which can be applied 'any blood test', for the purpose of determining whether the body of the arrested person 'bears any mark, characteristic or distinguishing feature, or shows any condition or appearance'.

It is submitted that the performance of blood group tests would fall within the scope of the contemplated amendment. However, the position in civil disputes would not be affected by this change in the law, and it is in civil disputes that the use of this evidence finds its greatest application.

In South Africa research into the complicated blood groups of Man is being intensified very energetically. Some of the rarer blood groups have already been discovered in this country and it should be possible today to have available all those antisera which are likely to assist the cause of justice.

It should be noted, however, that the performance of these tests requires knowledge, experience and skill exceeding those of the average medical practitioner and even of many a clinical pathologist. This work has passed beyond the field of the amateur, and we must today recognize that others than those medically qualified may be sufficiently expert to be of assistance to our Courts.

bekwaam genoeg vir hierdie werk. Voorlopig is daar nog geen wyse waarop enige burger deur die hof gedwing kan word om homself aan hierdie soort ondersoek te onderwerp nie; maar 'n belangwekkende afwyking word in die skild gevoer, t.o.v. gevalle van misdaad, by wyse van 'n amendement soos gepubliseer in die *Wysigingswetsontwerp op Kriminele Prosedure en Bewyslewering, 1950*. Indien die inhoud van hierdie Wetsontwerp wet word, mag van enige persoon, wat gevange geneem word oor enigiets, 'n bloedeksemplaar gevorder word waarop daar enige ‚bloedtoets' toegepas kan word ter vasstelling daarvan, of die liggaam van die gevangene ‚enige merk, kentekenende of onderskeidende eienskap besit, of enige omstandigheid of voorkome vertoon'. Dit word in die vooruitsig gestel dat die uitvoering van bloedgroeptoetse binne bestek van hierdie voorgenome amendement sal val. Hoe dit ookal mag wees, die posisie t.o.v. burgerlike geskille sal deur hierdie verandering in die wet nie beïnvloed word nie en dit is juis in die geval van burgerlike geskille waar hierdie bewysvoering sy vernaamste toepassing vind.

In Suid-Afrika word die navorsing oor die ingewikkelde bloedgroepe van die mens baie deurtastend verskerp. Sommige van die seldsamere bloedgroepe is alreeds ontdek geword in hierdie land en dit behoort vandag moontlik te wees om al daardie antisera beskikbaar te hê wat moontlik die saak van die gereg mag bevorder.

Daar moet egter op gelet word dat die uitvoering van hierdie toetse kennis, ondervinding en bekwaamheid verg, wat dié van die gemiddelde mediese praktisyn of selfs dié van menige kliniese patoloog oortref. Hierdie werk het die bestek van die beginner verlaat, sodat ons vandag dit moet erken dat ander persone dan medies gekwalifiseerdes, bedrewe genoeg mag wees om die howe te help.

SYNKINESES IN NEUROMUSCULAR RE-EDUCATION

IN INFANTILE CEREBRAL HEMIPLEGIA

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One of the most intriguing problems of neuromuscular re-education in infantile cerebral hemiplegia is the re-awakening of the cerebrally elicited function of skeletal muscles showing no (= zero) voluntary response. These muscles, often found in the 'spastic' syndrome, are flaccid. As the origin of this condition lies in the brain, the American pioneer of cerebral palsy, Dr. W. M. Phelps, named them 'zero-cerebral' (*abbreviation*: O.C.).

REVIEW OF METHODS OF NEUROMUSCULAR RE-EDUCATION OF O.C. MUSCLES

The well-known and widely-spread physiotherapist 'school' founded by Phelps treats this condition—according to Collis,[1] Egel,[2] and James[3]—with the help of 'automatic or confused motion'. This method, based on the principle of 'pathological overflow', side-steps the direct approach to volitional muscle contraction. Instead, it tries to elicit muscular contraction of a O.C. muscle by resisting the contraction of another muscle or muscle group which then passes on the impetus to the O.C. muscle. Thus a movement is produced by an automatic contraction. Hence the term 'automatic motion', while 'confused motion'—according to the terminology of the Phelps school—means that the contraction of the O.C. muscle is produced by way of a 'confusion' of an impetus which was directed towards a resisted muscle that is neurologically healthy. As not every muscle or muscle group, when resisted, passes on an impetus to the O.C. muscles which we wish to stimulate to contraction, the opinion is that one has to search very carefully all over the body for a muscle which 'does the trick'.

The first case of what came to be termed 'confused motion' was described by Phelps. It has since become the classical paradigm for this type of motion. Briefly stated, it would read: A spastic child with O.C. dorsiflexor muscles at the ankle was found by Phelps to accomplish dorsiflexion as soon as the homolateral hip flexion was resisted by the weight of a heavy box.

This is interesting in view of what Walshe,[4] citing Marie and Foix,[5] had written in connexion with hemiplegia as early as 1923, i.e. that although an isolated dorsiflexion of a foot may be impossible for a patient with some voluntary control over the affected leg, yet, if the patient flexes the whole limb, he cannot help automatically dorsiflexing also the foot.

To continue with the 'confused motion' method: as soon as some contraction of the O.C. muscle is achieved the physiotherapeutic idea is to reduce the resistance gradually until the mere thought of resistance suffices to produce a contraction of the O.C. muscle. However, this contraction remains always dependent upon the 'confused motion' (Egel [6]).

It must be mentioned here that although the contraction of the O.C. muscle is produced by resistance, it is not the term 'resisted motion' which is applied to this form of motion. This is reserved for the motion whereby the contraction of the very same muscle (a weak muscle) which should contract, is resisted, and it is not used for the contraction of a muscle different from the muscle which we wish to stimulate, as in 'confused motion'.

In contradistinction to the Phelps school, a different sort of 'resisted motion' has been recommended by Kabat and Knott [7] with the purpose of provoking a O.C. muscle contraction. Kabat and Knott approach even severely paralysed muscles *directly* and not by way of a detour via 'confused motion'. They recommend, *inter alia*, heavy resistance by the physiotherapist to the contraction of the flaccid muscle itself. This recommendation is based on the physiological fact that the amount of activity in a muscle, motor nerve or central motor pathway depends primarily on the percentage of the units being excited. Therefore the authors think that greater resistance to the contraction of a muscle should bring into play more motor units than with less or no resistance.

Another—and to my mind better—recommendation by the same authors postulates innervation of a muscle by voluntary activation of a more complex primitive pattern of which the muscle forms a part.

Fay [8] builds his system on the basis of a sound knowledge of postural reflexes, reactions of defence, tonic neck reflexes and pattern movements. To revert to Phelps' 'confused motion' example: Fay obtains the desired aim—*inter alia*—through the stimulation of the lower extremities by pinching, scratching, sudden passive flexion of the toes or lateral compression of the foot. This usually results in the leg being drawn up suddenly, flexing all the leg joints including hip, knee, ankle and toes (especially the hallux). This is a typical example of a 'defensive reflex' (Babinski), or a so-called 'reflex of spinal automatism'. Fay also makes an occasional remark about the possibility of initiating a crossed pattern movement by training the co-ordinated existing responses, e.g. of the right upper extremity, to aid the paralysed left upper extremity.

Walshe [9] gives an example of how hemiplegics sometimes even use *yawning* for exercising the fingers.

Besides these methods mentioned above, faradism is tried directly on O.C. muscles, but I have seen no proof of any positive result of this method. I also fail to see even a theoretical possibility of eliciting a *volitional* contraction of a O.C. muscle—for that is what we are aiming at—by faradism, as this type of current does not reach any cerebral motor centres at all. Certainly, as 'no electrical changes occur in the spastic type' of muscle (Wallin [10]), faradic current might—in favourable circumstances—provoke a contraction of the O.C. muscle thus hampering muscular atrophy through inactivity. But as a rule even this is not the case, as spastic muscles in the vicinity, being hyper-irritable, catch, as it were, and respond to aberrant currents much more easily than the O.C. muscle catches and responds to the main current. All we get, therefore, is an unwanted contraction of a spastic muscle.

Lucas [11] thinks that faradic stimulation of flaccid muscles might facilitate the pathways and encourage the patient.

I have attained the best practical results in eliciting voluntary contraction of O.C. muscles by means of the 'confused motion' method, but that only of the very same type whereby Phelps found the principle of 'confused motion'. I also found it with 'synkineses'.

THE SYNKINETIC METHOD

The word 'synkinesis' is derived from the Greek word συν = *with*, and κινησις = *movement*, thus literally meaning a concomitant or associated movement. In the narrower sense in which it is used here, it means an unintentional contralateral and mirrorlike-symmetrical movement elicited by a volitional movement starting from the unaffected side. It is the sort of synkinesis which is termed 'spasmodic synkinesis'. Spasmodic synkineses are characteristic for pyramidal lesions (Walshe,[12] Krey [13]). Also Schaltenbrand,[14] regarding infantile cerebral palsy, states: 'The inclination to symmetric synkineses of the affected side is characteristic. They cannot voluntarily be suppressed by the patient.'

When I treated soldiers suffering from cortical pyramidal lesions resulting from bullet wounds, I frequently found with hemiplegics that if the healthy limb practised simultaneously with the paralysed one, nervous muscle response was sooner attained than by trying to make the affected limb move independently. Although this was a somewhat widely known observation it appeared not to have been perceived as a treatment by synkineses. Therefore it was simply said [15] that the healthy side 'shows' the paralysed side how to do it. Sometimes movements of both sides were attempted simultaneously [16] but often 'the healthy side showed the required movement first, whereupon the paralysed one had the task to copy it'.

I also found in orthopaedic gymnastics that neurologically healthy children, on being asked to spread the toes (widely forgotten skill) cannot do it. Yet if you ask them to spread the fingers simultaneously, the toes soon show the first signs of the movement.

When after this experience I once more came into touch with the physiotherapeutic treatment of infantile cerebral hemiplegics, it immediately struck me how well (and even better) a treatment on the basis of synkineses worked with children suffering from infantile cerebral hemiplegia, compared with adults suffering from a similar upper motor neurone lesion acquired in later life. This observation is stressed by R. M. Steward.[17]

Some children, when asked to do a certain movement

by a O.C. or a cerebrally very weak muscle, accomplish the task by doing the same movement contralaterally and, when asked why they do so, if they are intelligent they answer they had noticed that 'otherwise it is impossible'. This was also observed by Fischel,[18] the mother of two spastic boys. She appears to have made use of this for treating her first-born boy, and she remarks: 'This aided a great deal.'

In my experience dorsiflexion of the foot or hand, abduction of the thumb, opposition of the second finger to the thumb and of the fifth finger to the thumb respond comparatively readily to synkinetic treatment.

Although the synkinetic method works more easily with hemiplegic patients, it can also be used with paraplegics. This happens because synkineses also link hands and feet—only to a slighter degree (for in an ontogenetically older stratum the human being is a quadruped).

Although synkineses might work without further aid, they appear to be still more facilitated if one gives resistance to the movement of the *healthy* side. So, e.g. if I wish to attain a movement of the right foot by the O.C. dorsiflexors, I ask the patient to try to dorsiflex both feet while I resist the motion of the healthy side manually. If then the patient tries hard for a little while (not with a jerk but perseveringly) to push up my hand with the healthy foot, he will not only succeed in doing so, but (after a short interval) an initial tonic contraction of the dorsiflexors at the paralysed side will as a rule be observed.

This again is a sort of 'resisted motion', but it is not, as with Kabat and Knott, the paralysed motion itself which is resisted, but the contralateral intact one. Walshe[19] even thinks that 'force' is essential for these synkineses; and I fully agree with what he says further[20]: 'The adequate stimulus is proprioceptive and of some duration'.

To resist muscular contraction manually or by a heavy load is only the simplest and most obvious form of resistance, for even tiredness must be counted as a sort of resistance. In fact it helps to elicit the contraction of a contralateral O.C. muscle. As early as 1906, Curschmann[21] wrote that under certain conditions, e.g. tiredness because of protracted examination or because of volitional movement with heavy loads, he succeeded in provoking strictly symmetrical contralateral synkineses with practically all *normal* persons. He adds: 'The important factor for the eliciting of contralateral synkineses is the amount of energy required at the volitional act.' Also Kabat's and Knott's plea for 'disregarding fatigue' seems to me to point towards the same end.

These spasmodic synkineses facilitated by heavy resistance are, moreover, modified by the various neck, optic and labyrinthine reflexes. A short time ago Yamshon, Machek and Covalt,[22] based on Magnus' and de Kleyn's work, tested the influence of the tonic neck reflexes on 22 patients with hemiplegia due to a cerebral vascular accident. They found that this type of patient can extend his paralysed elbow to a greater degree and with greater strength when the face is turned towards the involved side. Schaltenbrand's[23] findings, based on myography, confirm this.

One must never lose sight of the modifying influence of these various reflexes mentioned above; moreover, one must be constantly on the look-out for them, in order to make use of them therapeutically.

THEORETICAL FOUNDATION OF THE SYNKINETIC TREATMENT

In cerebral hemiplegia the affected skeletal muscles are no longer volitionally ruled by the contralateral cortical motor neurone. Yet in the affected extremities many spontaneous reflex pattern movements remain, which are similar to the movements noted by Sherrington in the decerebrate animals. Their origin lies 'in the lower levels of the midbrain, the medulla and the upper cervical and thoracic portions of the cord' (Fay[24]). Synkineses in a broader sense are regarded by Walshe[25] as released postural reactions, because the normal postural adjustments of the skeletal musculature with voluntary purposeful movements are 'necessarily bilateral and widespread' in forceful movements. The (unaffected) brain-stem is regarded as the place of origin of postural adaptation. This, however, would only explain complex movements on a *lower* motor neurone level. Perhaps it also explains why resistance to muscular contractions (forceful movements) facilitates the contraction of O.C. muscles.

Recent writers like Krey[26] think that the origin of the spasmodic synkinetic impulses lies in the same pyramidal area which produces volitional impulses, thus in the unaffected cortex. These synkinetic impulses are then conducted to the affected skeletal muscles and that most probably by means of the ipsilateral pyramidal fibres.

One must bear in mind that once there existed a *primary bilateral innervation* (Krey[27]) and that this becomes only latent (Krey[28]) when the child—unconsciously—learns to avoid purposeless co-innervations by inhibiting them.

Well-known experiments with monkeys have proved that with total excision of one motor cortex arm centre, the contralateral arm could, although lacking finer skill, still be used if the healthy arm was eliminated by tying up (note: resistance) or by amputation; also the results of hemispherectomy in human beings are a proof for bilateral innervation.

Now it is this inhibition, the place of origin of which is believed to be the cerebral cortex, which again must be eliminated and the suppressed innate synkineses must be revived. Then by directing the inhibition to the contralateral movements a volitional ipsilateral movement is learnt and disused old functional pathways are re-opened. The fact that this is possible leads us to assume that in the motor cortex of each hemisphere there are separate motor neurones for the contralateral and for the ipsilateral side, and not that both sides are governed by one and the same motor neurone. That the contralateral and the ipsilateral motor neurone are anatomically close to each other is highly probable.

CONCLUSIONS

From the data obtained I believe that this method of approach to contraction of O.C. muscles or cerebral weak muscles by means of synkineses, although an 'indirect method of teaching' is of so much value in the neuromuscular re-education of infantile cerebral hemiplegics that it should no longer be omitted in their training programme.

However, as this method is also tedious and needs great patience and much time on the part of the physiotherapist as well as the patient, it would be an enormous step forward if we could avoid this slow process by such an

operation as the removal of one cerebral hemisphere described in 1950 by Krynauw.[29] This operation is the more important because it not only lessens spasticity and clumsiness but improves also personality, behaviour and mentality.[30] This operation has suddenly brought a South African to the leading ranks of cerepalsists of the world.

SUMMARY

The foremost attempts to neuromuscular re-education in infantile cerebral hemiplegia are critically evaluated. They are: Phelps' method of 'confused motion', Kabat's and Knott's method of direct resisted motion and also of complex primitive pattern movement, Fay's method of defensive reflexes, and the still widely adapted faradic treatment.

Attention is drawn to the presence of spasmodic synkineses in infantile cerebral hemiplegia. It is maintained that they can be used to elicit volitional contractions of zero-cerebral or weak cerebral muscles. It is further shown that resistance to muscular contraction on the healthy side is the main reinforcing factor for these synkineses. Neck, optic and labyrinthine reflexes are of less importance.

Finally a theoretical foundation of the synkinetic treatment is offered.

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ASPECTS OF CRANIO-CEREBRAL TRAUMA

4. CLINICAL FACTORS*

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The clinical factors will be discussed only insofar as they are indicative of grave injury, of conditions amenable to therapy, or of recognition of signs which, if neglected in their portent, may lead to such deterioration that subsequent treatment is unable to avert a fatal issue.

In concussion *per se*, if death does not ensue immediately (a rare event), recovery will invariably occur. Concussion, however, must always be regarded as a diagnosis made in retrospect, when the subsequent recovery has ruled out the development of other pathological manifestations; but even then the occasional development of the chronic subdural syndrome is always a dangerous possibility. By far the commonest causes of death are contusion and laceration, with a variable amount of added oedema, but here again recognition is not so simple a matter as commonly taught. Severe localized contusion can occur without any impairment of consciousness or cerebration, such cases being almost always associated with an overlying fracture. Evidence of organic destruction is immediate and persists permanently though some improvement will often take place after the subsidence of oedema. These patients, with proper surgical treatment, usually have an excellent immediate prognosis.

Not so, however, the case that comes in unconscious or has a history of prolonged unconsciousness. There may or may not be external wounds or fracture of the skull. These cases are almost always associated with contusion or laceration of the brain as shown in the vast majority that fail to survive. Clinically, in the moderately severe cases, there is a certain amount of cerebral irritation or traumatic delirium. The patient lies on his side, curled up in bed with the blankets pulled up over his head (photophobia), is restless, often violent, micturates in his bed, often refuses to eat or drink and is generally unco-operative. The temperature is usually moderately raised, but the pulse, respiration or blood pressure show no characteristic change. Lumbar puncture reveals a variably blood-stained fluid of normal or moderately raised

* The References will be published at the end of the concluding paper in this series.

pressure. Bagley (1941) states that it is important to recognize the irritative low intracranial pressure types. Provided no complications such as meningitis or haematoma-formation occur, the prognosis in the above syndrome is good, as far as life is concerned.

Another group comprises those patients who have obviously sustained a severe injury, as shown by scalp and orificial bleeding, and are stuporose or deeply unconscious. Alcohol is often a dangerously deceptive superadded factor. In the comatose patient no reaction to painful stimuli is elicited; the breathing, which may be normal, is usually deep and stertorous; the pupils are usually moderately dilated and react sluggishly to light; later, in the deteriorating patient, becoming fixed and dilated; the corneal reflex disappears; the tendon reflexes are absent or minimal; the plantar responses equivocal and the limbs flaccid. The temperature, pulse, blood pressure and cerebrospinal fluid pressure, as discussed previously, show no characteristic change, though the latter, blood-stained, may be considerably elevated, but may often fall within the range of normality. Rarely does the breathing assume a Cheyne-Stokes rhythm; the temperature may rise to 104-106°, though this is a terminal event. It must be emphasized, however, that there is a type of patient not deeply unconscious, i.e. he is rousable but lapses back into coma, and displaying to a lesser degree the clinical manifestations enumerated above, who has just as severe a degree of cerebral trauma, as demonstrated at autopsy. The patient remains in his state of 'rousability' practically up to the time of death, in contradistinction to the gradual state of deterioration usually observed.

The third group comprises all those conditions usually classified as 'cerebral compression' and generally described as an inexorable condition exhibiting classical manifestations. The classical syndrome is *not frequent* and awaiting its development or diagnosing only on its presence will result in fatalities that could possibly have been averted.

Oedema of the brain, besides its effect of increasing the volume of the intracranial contents, may have local effects often indistinguishable from that of a localized contusion. Transient aphasia, paralysis or paresis, associated with a clear cerebrospinal fluid under a normal or slightly increased pressure, recovering without sequelae, may be regarded as being evidence of local oedema or general oedema with local effects. In a number of cases explored for a possible haematoma, craniotomy revealed no evidence of haemorrhage or cortical injury. It is true that a subcortical haematoma, ecchymosis or petechial haemorrhages with surrounding oedema may give a similar clinical picture, but resolution would take weeks rather than days to occur.

Cerebral oedema can also present with the syndrome of cerebral irritation, particularly, as so frequently happens, when it is associated with other forms of cerebral trauma. Oedema may, however, be the *only* pathological manifestation of head injury. It can, *per se*, exhibit all the clinico-pathological effects of a space-occupying lesion, including the interval syndrome, and we suspect that it may give rise to epileptiform convulsions similar to those occuring in other forms of cerebral oedema, as, e.g. in uraemia.

Acute Subdural Haematoma. In discussing the pathology, stress was laid on the fact that contusion or laceration of the brain is almost invariably associated. Clinically the same lack of distinction prevails. Although a so-called lucid interval may occur, in most cases the two conditions so overlap and influence each other that a deteriorating state of the patient is the only positive indication of increasing intracranial mischief. The patient usually has a pronounced disturbance of consciousness, increasing the difficulty of evaluating any focal signs which, in any case, will be meagre. Paralysis when contralateral is important but may be ipsilateral when coning is occurring. Unilateral dilatation of the pupils in a stuporose or comatose patient is a sign of great urgency in treatment, indicating commencing or even advanced herniation. The cerebrospinal fluid may theoretically be clear, but in practice there is always a variable bloody discoloration due to associated cortical damage. The pressure may be high but can be normal as shown in one of our cases (Figs. 7 and 8, this *Journal*, 6 October 1951) where the blood-stained fluid was under a pressure of 150 mm. fluid. Yet on opening the dura the subjacent blood was under such great tension that it spurted out, as if an artery had been severed. Bradycardia may occur and is important when associated with deepening stupor.

In an analysis of 30 fatal cases of acute subdural haematomata, fracture of the skull was found present in 70%, with involvement of the base in 60%. Bleeding from the ear and nose was by no means infrequent. The haematomata, in this series, were bilateral in 80%.

The Subacute Subdural Haematoma. In the acute phase the manifestations are profound and death usually results in the first two to three days. When there is retrogression after this period, or signs become manifest then, we are dealing with the subacute form of bleeding. Increasing drowsiness leading into stupor and coma is the most important clinical finding though it may be preceded by headache, often of great severity, and 'conduct or personality changes'. Paralysis, often on the 'wrong side' and pupillary changes are again important findings. Changes in the optic discs may occur but are, as in all acute traumatic space-occupying lesions, rare.

The Chronic Subdural Haematoma. The symptoms usually come on within the first three to four months, but may only manifest themselves much later. Headache, drowsiness leading to stupor, nausea and vomiting, papilloedema (unusual), pupillary changes, paresis or paralysis are the usual clinical findings. An important, almost characteristic sign is the personality change that occurs, amounting sometimes to severe mental disturbance; but of greater significance is the fluctuation of these changes, the so-called 'in and out' state (Foster Kennedy), the patient having episodes of acute personality changes. It is important for practitioners to be 'haematoma conscious'.

The subdural hygroma clinically may resemble all the varieties of subdural haematomata or can present as a classical 'post-traumatic syndrome'. It is infrequently associated with an increased cerebrospinal fluid pressure. In infants there may be gradual enlargement of the head with prominent scalp veins. Poor feeding, fretfulness and convulsions are frequent findings.

Extradural Haemorrhage. Classically the patient suffers a blow to the head, is concussed for a few minutes or

even longer, for an hour or more, regains consciousness and, after a variable period of hours to days, again lapses into unconsciousness, the interval between the two phases of coma being known as the 'lucid interval'. Unilateral paralysis, or paresis, the development of the classical Hutchinson's pupil, bradycardia and a raised blood pressure with an increase in the cerebrospinal fluid pressure complete the typical picture.

Although King (1943) found 50% of extradural haematomata to fall in the above class, observers like Munro and Maltby (1941), Gurdjian and Webster (1948) and Browder and Meyers (1936) found the classical picture to be rare. A rapid meningeal haemorrhage may be so superimposed on the primary 'concussive' phase as to result in the complete absence of a 'lucid interval', the unconsciousness of the concussion merging into that of compression. This has particularly been the experience in Bantu patients. Of special importance is the evidence of swelling and bogginess of the scalp, especially of the temporal regions, due to the escape of blood via an underlying fracture into the subaponeurotic space. A contralateral paralysed limb in this stage frequently is flaccid compared with its fellow, becoming spastic only after some time. An advancing paralysis of the face, upper limb and lower limb or Jacksonian fits in this sequence is strongly indicative of a spreading extradural haemorrhage. The reflexes are of some value, particularly a positive Babinski sign. In general, however, all signs are equivocal except those of increasing or persistent stupor and pupillary changes. In extradural cerebellar haematoma the neurological findings again may be meagre. Owing to the fact that the haemorrhage is often venous from injury of the lateral sinuses, there may be a long lucid interval. Signs suggesting pressure on the cerebellum in the posterior fossa are neck rigidity, papilloedema, nystagmus, reduction or loss of the deep reflexes, tonic convulsions, ataxia and hypotonia, of which the latter two are the most consistent and important (Anderson, 1949).

In a series of 23 fatal cases studied retrospectively, it was found that no case was admitted to hospital in a conscious state though a history of a lucid interval was obtained in four.

Subcortical haematomata cannot be distinguished clinically from subdural or extradural varieties, giving the same march of symptoms and signs. Hence the necessity of needling the brain, particularly the frontal and temporoparietal regions, at the time of exploration with negative findings. Air studies and angiography may be essential.

Intraventricular Haemorrhage. As this is almost always secondary to a rupture of a subcortical haematoma, the clinical picture is that of a sudden deterioration of a previously serious state. Rigidity of all the limbs may be present but we have found, experimentally and clinically, that a massive rise in the blood pressure with a rapidly deteriorating state is almost pathognomonic; in one case the rise of blood pressure reached over 300 mm. Hg *at the time immediately preceding death.*

The Interval Syndrome. This is generally associated, in the minds of most practitioners, with extradural bleeding, though it can occur as frequently in the acute and subacute subdural varieties as in the extradural (Gurdjian and Webster, 1948) and may last for hours, days or weeks. Owing to the slower expansion of the subdural haemorrhage the 'physiological events' are more prolonged. Although in these cases the prognosis improves with the length of the interval, the opposite applies in the extradural haemorrhage (Mackenzie, 1939), where, with a long latent interval, in spite of removal of the clot, the patient does not recover.

Intracortical haemorrhages can present a similar picture either in the acute phase or in the late result (e.g. the so-called 'traumatic apoplexy'). Frequently the haemorrhage is a secondary phenomenon, being part of a large contusion and, though small, it may dissect into the centrum (Courville, 1945). In other cases again, where the syndrome presented itself, either operation or autopsy revealed diffuse brain injury in the nature of contusion, laceration or oedema. In 10 cases with a lucid interval, Gurdjian, Webster and Arnkoff (1943) found that four were due to subdural or extradural haemorrhage, four were due to diffuse brain injury and two to fat embolism. It is because of the doubtful nature of the causative lesion that Browder and Turney (1942) have resorted to the use of air studies in practically all their cases.

It will be understood, of course, that the 'lucid interval' is not free from symptoms. The patient complains of headache, giddiness, irritability, vomiting and, still later, exhibits drowsiness from which he is rousable at first, gradually lapsing into complete coma. The reason for the latent period is usually given as due to shock, in which the lowering of blood pressure causes a cessation or diminution of bleeding which subsequently, on recovery, is reactivated. Shock, however, is not an important factor in head injuries and a more rational explanation (Rowbotham, 1949) is that the brain can initially accommodate itself to a space-occupying lesion and it is when the brain loses this capacity that the second phase of coma ensues.

A further important type of sequence to be borne in mind is that occurring in acute alcoholism. Here the coma of concussion is fused with that due to alcohol, the latter phase, initially superimposed, eventually superseding the former. The phase of alcoholic coma may then merge into that of compression, the whole all too frequently being diagnosed merely as 'drunkenness'.

The Clinical Evidence of Cerebral Compression. In the section on surgical physiology it was pointed out that the *usually accepted* clinical indications of compression are unusual and often deceptive, and though it is well to remember Kocher's postulates as indicating the classical pattern of events, in practice there is such a pathological admixture that awaiting the march of symptoms and signs, even if they be present, is to spell the doom of the patient. Unfortunately our standard of measurement of the clinical condition and progress of the patient is at fault. The more experience one has of these severe cranio-cerebral cases, the more one is forced to the conclusion that there is no criterion for measuring the acute intracranial changes occurring in them.

The two clinical findings of most importance are the state of consciousness and the condition of the pupils. It is our opinion that any head injury in which the stupor or coma persists for more than 12 hours, irrespective of any or all the associated clinical findings, irrespective of whether slight improvement is occurring (often a fallacious impression), must be submitted to the final diagnostic

procedure of exploratory craniotomy, or, in experienced hands, ventriculography. The latter may be indicated in cases of continued coma when exploration has not revealed any suspected collection of blood.

In assessing a patient's state of consciousness great clinical judgment is required, particularly in recognizing a deteriorating state. The doctor must literally live at the bedside of the patient. The latter should actually be roused every hour, in addition to hourly recordings of the pulse-rate, blood pressure, respiration, temperature and pupil sizes, any or all of which may give confirmatory evidence of deterioration. The onset of compression can be extremely rapid—instead of watchful inactivity, the practitioner may inactively watch the whole 'march of events' to the mortuary table.

Pneumo-Encephalography. Of the two methods, spinal and ventricular, the former is interdicted in the acute case showing evidence of increased intracranial pressure, e.g. prolonged unconsciousness or a dilated pupil. These cases may have a greatly increased supraforaminal or supratentorial pressure and spinal drainage, even with air replacement, may precipitate a fatal issue. In the subacute and chronic subdural haematomata Gurdjian and Webster (1948) have found encephalograms very helpful and well tolerated. There may be a midline ventricular shift, or absence of cortical air markings on the affected side. An almost pathognomonic sign is the finding of a collection of air medial to the haematoma. Ventriculography, in experienced hands, can have no valid contra-indication and may not only be diagnostic as an air study, but in its performance collections of blood may be encountered. Furthermore, by relieving an internal hydrocephalus it may reduce the internal herniation so often causing it.

Electro-Encephalography, in conjunction with clinical study, may be an aid of some value, particularly in the subacute and chronic lesions.

Roentgenological Examination. In acute cranio-cerebral trauma, in a seriously ill, very restless or even shocked patient, the procedure may do more harm than good, except in two classes of cases. Firstly, when an extradural haemorrhage is suspected it is almost imperative to obtain what we term a 'scout' film of the skull, as the vast majority of these cases are associated with a fracture in the immediate vicinity of the haemorrhage. However, fracture may be absent, particularly when the bleeding is venous in origin (Reichert and Morrissey, 1941). Localization of the fracture line across the middle meningeal vascular markings is strong presumptive evidence of damage to these vessels, in cases where the clinical findings are compatible with such a diagnosis. Secondly, in compound fractures a preliminary radiological investigation may reveal the extent of the bony damage and the amount and degree of depression of fragments. Slight depression of the outer table may be associated with marked inner table displacement.

PACHYDERMOPERIOSTOSIS

THE SYNDROME OF TOURAINE, SOLENTE AND GOLÉ

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This disease was recognized by Friedreich[9] in 1868, but it is only in the last 20 years that its peculiarities have become better known and its individuality firmly established.[3, 5, 10]

The name pachydermoperiostosis—the term favoured by Jean Vague[11]—indicates the main features: a thickening of the skin and the bones. Skin thickening is chiefly to be seen in the scalp, upper eyelids, palms and fingers. Bone thickening is usually noted in the distal portions of the limbs, particularly in the tubular bones.

The disease is confined to males, and has been noted on several occasions in brothers or the children of blood relations. It therefore has hereditary features but the manner of inheritance is not fully known. Typically the abnormal processes begin just after adolescence and progress slowly, smoothly and painlessly for several years. Natural and lasting arrest may then occur. Most of the cases reported, including ours, developed only as far as this stage. But after a variable interval an increase in bone production may begin again. It may then develop towards a universal hyperostosis of the skeleton with ossification of ligaments and fusion of certain joints. Joint fusion is seen especially in the carpus, the tarsus and the vertebral column. Eventually the vertebral hyperostosis may cause compression of the spinal cord and nerve roots.

Characteristically the disease is unassociated with any constant abnormalities of the endocrine glands or viscera. There is also no doubt that mild forms of the condition occur in which either the skin or bone changes are relatively insignificant.

One of the manifestations of the skin hypertrophy in this disease is the presence of clubbed fingers with watch-glass nails. For this reason some of the milder cases of this disorder have been regarded as varieties of Pierre Marie's *ostéo-arthropathie hypertrophiante pneumique*, in which the precipitating factor, whether pulmonary or otherwise, was lacking. Furthermore, the hypertrophy of

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bones and skin in the extremities has also led to the mistaken diagnosis of acromegaly, although the bones themselves are in fact only broader without being increased in length. Some have tried to make a diagnostic compromise between these two conditions by speaking of an acromegaloid variety of hypertrophic pulmonary osteoarthropathy.

Actually it appears that pachydermoperiostosis is sufficiently distinctive and has been observed frequently enough to make it an entity in its own right and not merely a variant of some other disease. Table I illustrates

10 years before admission his hands and feet began to increase painlessly in size. This condition is now arrested. For the past few years he has been apt to develop orthostatic oedema and pain in the knees after a day on his feet. Three years ago the patient alleges that he experienced a general swelling of the body and eyelids which soon disappeared. The cause of this is obscure. He has also had an occasional cough with tightness in the chest.

He has no children. His parents, a brother and a sister are all said to be normal. No other facts of importance were elicited in the history.

TABLE I

	Acromegaly	*Pachydermoperiostosis*	*Hypertrophic Pulmonary Osteoarthropathy*	*'Thyroid' Acropachy*
History and Symptoms ..	—	Painless. Steadily progressive	Painful. Progress in recurrent attacks	Often a history of hyperthyroidism and thyroidectomy
Age of onset		Onset in males aged 15–20	Males and females.	Adult males.
Sex incidence	—		Usually older than in pachydermoperiostosis.	All ages.
Family history		Familial features	Not familial.	Not known to be familial.
Skin changes	Hypertrophy of skin on scalp may be present. General skin hypertrophy. Large tongue.	Hypertrophy of skin on scalp, eyelids, palms, etc. Skin elsewhere normal. Tongue normal. Clubbed digits.	No thick skin. Clubbed digits.	'Malignant' exophthalmos. Possibly pretibial myxoedema. Clubbed digits.
Internal changes	Pituitary tumour.	Thyroid and pituitary fossa normal. B.M.R. usually normal.	Disease of lungs, heart, etc.	B.M.R. low or normal.
Bone changes	Mandible and short tubular bones lengthened. Tufting of terminal phalanges. Exostoses may appear. At fused epiphyses endosteal and periosteal growth particularly apparent. All bones may show changes.	Mandible normal. Bones not increased in length. Remodelling of entire bone structure with hyperostosis and reduction of marrow cavity. Lamellar fusion of new bone on surface of old cortex. May progress to involve whole skeleton. Terminal phalanges usually relatively unaltered.	Mandible normal. Bones not increased in length. Pure subperiosteal inflammatory deposition of new bone. New cortical bone in rings. Soft tissue layer between this and old cortex. Never universal. Terminal phalanges usually tufted.	Subperiosteal new bone in hands. Terminal phalanges may be tufted.
Joints	Hypertrophic and degenerative changes.	Usually unaffected. Exostoses may secondarily affect joints. Bony fusion in advanced cases.	Chronic synovitis. No ankylosis.	

significant points in the differential diagnosis, and includes reference to 'thyroid' acropachy,[7] which presents some resemblances towards this group.

Interest in pachydermoperiostosis seems to have been greatest in Europe, to judge by the number of contributions. Cases are also reported from the Americas,[3] but as far as we know the disorder has not been described before in a South African Native.

CASE REPORT

The patient was a 30-year-old Bantu male referred to the Pretoria Hospital from the country with the provisional diagnosis of acromegaly. He was admitted for investigation to Dr. H. W. Snyman's clinic.

He gave a poor history, but it appeared certain that

Examination of the *external features* revealed the following: His expression (Fig. I) was woebegone, like that of the classical mask of tragedy. Permanent V-shaped furrows creased the forehead and pointed down to the root of the nose. These were made even more marked by constant tension on the brow to counteract a ptosis of the upper eyelids. The redundant skin folds between the creases on the forehead were not specially thick or seborrhoeic. The ptosis was due to a heavy thickening of the lids with an excess of skin forming a row of folds at the upper edge of the eye socket. There was slight drooping of the lower lids as well.

There was no excess of skin on the hairy scalp. The anterior hair line at the top of the forehead was concave downwards with complete absence of temporal recession.

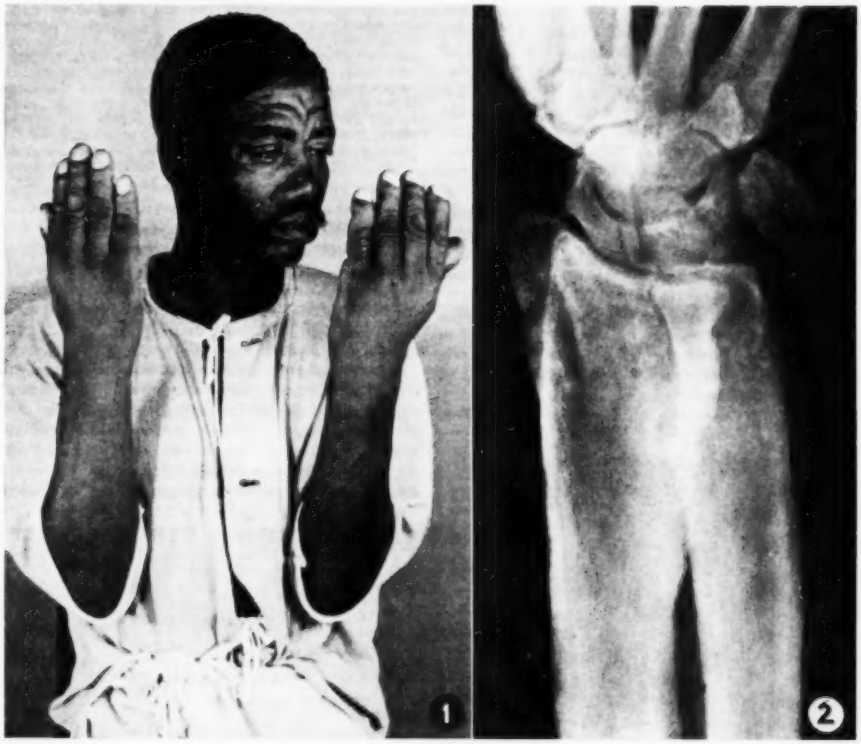

Fig. 1. Pachydermoperiostosis. The furrowed brow, heavy eyelids, broad wrists and hands with clubbed fingers can be seen.
Fig. 2. Radius and ulna showing hyperostosis and osteophytosis. Pisiform also affected.

This feature does not appear to have been specially noted, but it can be seen in all the illustrations collected by Touraine, Solente and Golé.[9] The patient had long eyebrow and eyelash hairs, and a small moustache and beard. In general his facial skin was not hypertrophic but rather smooth and delicate, with slight increase of pigment. Chin, ears and lips were normal. The whole face was broadened by very large bilateral chronic parotid swellings, presumably of the familial or racial type, as discussed by Castaigne.[2]

There was a striking breadth of wrists and palms with thickening of the fingers. The circumference of each wrist over the distal ends of radius and ulna was 20 cm., and the circumference of the middle finger round the proximal interphalangeal joint was 8 cm. The skin over the fingers was loose, velvety and pigmented with a little acanthotic increase over the knuckles. At the base of each nail the skin was infiltrated, the nail cuticle thickened, and the nail plates exhibited a watch-glass deformity without dystrophy. At the metacarpophalangeal and proximal interphalangeal joints there was some hyperextensibility.

The palms were fairly mobile and diffusely thickened. There was no hyperidrosis, pigmentation or hardening of the palms. The normal folds were deep and the hypertrophied skin formed numerous extra folds, especially across the hypothenar eminence. The dorsa of the hands were pale, with smooth, thin, mobile and velvety skin showing a slight warty yet mobile (acanthotic) hypertrophy at the sites of old injuries.

The feet were warm and not hyperidrotic. The terminal phalanges of the inner toes especially were clubbed, with thick, broad, oedematous, slightly erythematous tissue at the base of each nail. The nail cuticles were thick and the nail plates thick and broad, with torn, asbestos-like under-surfaces at the free edge. Velvety skin hypertrophy with pigmentation extended especially over the outer toes. There was intertriginous sepsis between all toes except the first and second.

There was a diffuse fissured hyperkeratosis of the

pressure points of the soles and sides of the feet, but the skin of the instep was pliable and not hyperkeratotic. Like the backs of the hands, the dorsa of the feet showed patchy islands of dark mammillated epidermal hypertrophy.

The left and right ankles measured respectively 28 and 30 cm. in circumference just above the malleoli. This thickening was all deep and bony.

In general the skin showed a remarkable softness, smoothness, delicacy of texture and mobility, without hyperelasticity or formation of subcutaneous nodules. Axillary hair was sparse and pubic hair normal; there was no stout hair anywhere on the trunk. There was also no flexural acanthosis. He was in a good nutritional state without being fat.

A portion of the hypertrophic skin of the forehead was excised for histological study. For the preparation and examination of the sections we are indebted to Prof. C. Jackson. The findings were as follows:

The epidermis and its appendages are unchanged, and there is no evidence of hyperplasia of the sebaceous glands. There is considerable increase in collagen and elastic tissue throughout the corium, and in the midcorium there is basophilic degeneration of the elastic tissue with slight clumping but no fragmentation of the fibres. Reticulum stains reveal no abnormalities. Throughout the corium there are small knots and bands of perivascular infiltrate, consisting particularly of monocytes and lymphocytes. Mast cells are also considerably increased, and there is evidence of fibroblastic proliferation. In the midcorium the lymphatic plexuses are prominent. In these channels and in the venules, accumulations of haemofuscin pigment are to be seen in the cells of the endothelium. There is no oedema or mucinous infiltrate.

There was no limitation of movement in his joints. The elbows and knees were rounded and prominent. His gait was made clumsy by his heavy legs which he lifted only slightly from the ground while walking and advanced by circumduction. He was 160 cm. in height; span, 165 cm.; the lower measurement (symphysis pubis to floor) 81 cm. Serum calcium, 11.2 mg. per 100 ml., serum phosphorus 2.65 mg. per 100 ml., alkaline phosphatase 6 King and Armstrong units.

The entire skeleton was examined radiologically. The abnormal change noted in the tubular bones was a diffuse thickening of the cortex with greatly increased density of the bone; there was little or no visible distinction between the old shaft and the newer periosteal and endosteal deposits. This hyperostosis resulted in narrowing of the marrow cavity, and was in places clearly laminated in the

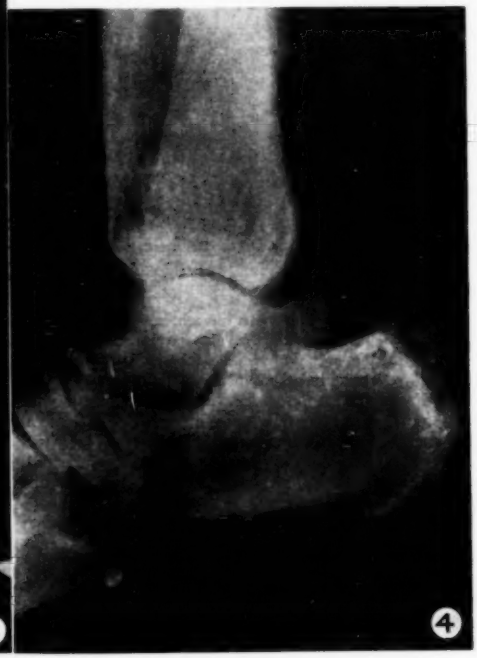

Fig. 3. Hand showing characteristic barrel shape of short tubular bones due to hyperostosis.
Fig. 4. Ankle region and tarsus with gross hyperostosis and osteophyte formation. Involvement of the tarsus as seen here is found only in advanced cases.

long axis of the bone. These changes resulted in a conspicuous widening of the bony shaft giving a more cylindrical shape to the tapered bones and a barrel-shape to some of the short tubular bones (Fig. 2). The affection was symmetrical, and involved all the long bones of the upper and the lower limbs, but was more evident distally. In the tubular bones of the hands and the feet there was maximum evidence of this change in the proximal phalanges of the hands (Fig. 3), the first and fifth metatarsals and the proximal phalanges of the big toes.

Osteophytic growths were also conspicuous on the surfaces of the enlarged bones, especially in the neighbourhood of the elbows, wrists, knees and ankles (Fig. 4). They were slightly in evidence as tufts on the terminal phalanges of fingers and toes, and also affected the following bones of the carpus and the tarsus: pisiform, calcaneus, talus, navicular and first cuneiform (Figs. 2 and 4). These osteophytic growths were also seen along the interosseous membranes of the forearms, the lateral edges of the scapulae and the anterior margins of the ilia.

The skull and the sella turcica, vertebral column, ribs, sternum and clavicles were normal. There was no abnormality of the joints.

The lungs were normal clinically but for an occasional rhonchus. Skiagrams showed no disease of the lungs. The heart was normal clinically, and the arm-tongue circulation time was within normal limits (Decholin, 12 seconds). The blood pressure was 130/75 mm. Hg. Skiagrams showed slight left ventricular enlargement. On the electrocardiogram Dr. A. J. Brink reported:

Sinus rhythm, regular. P not abnormal. P-R 0.20 second. Q-T_c 0.40 second. No axis deviation. Heart is in a semi-vertical position with some clockwise rotation. ST segment not depressed. T wave sharply inverted in I, II, III, V_4–V_6, V_2 and VF. Biphasic T in V_3. *Comment*: Left ventricular hypertrophy or strain in a semi-vertical heart.

The gums were infected. Gastric analysis showed a lowered value for total acid but was otherwise normal. The liver was not enlarged, and function tests were as follows: total protein 6.78, albumin 3.37 and globulin 3.41 grammes per 100 ml.; cholesterol 153 mg. per 100 ml.; total bilirubin 0.7 mg. per 100 ml.; van den Bergh, direct negative; thymol turbidity 6.5; thymol flocculation, nil; serum colloidal gold, 3. The total blood ascorbic acid was 0.29 mg. per 100 ml.

The central nervous system, blood count, urine examination and sedimentation rate were normal. An intravenous pyelogram was normal, and no calculi were revealed. The Wassermann reaction was negative.

There was no clinical abnormality of the thyroid gland. There was no exophthalmos. The basal metabolic rate was −28%, but the circumstances of the test were not entirely satisfactory. There had been no loss of libido or potency.

General examination revealed nothing further of note.

DISCUSSION

The nomenclature of this disease is still unsettled and a great many titles have been suggested for it on grounds of priority, accuracy (real or alleged) and descriptive completeness. This matter has been fully discussed by Uehlinger,[10] who adduces a number of arguments for and against titles such as the following: Friedreich-Erb-Arnold syndrome; megalia cutis et ossium (Grönberg [5]); generalized osteophytosis; pachydermie plicaturée avec pachyperiostose des extrémités (Touraine, Solente and Golé [9]); Hyperostosis generalisata mit Pachydermie (Uehlinger). We have chosen the term pachydermoperiostosis because of its relative brevity and accuracy; it appears, moreover, that the disease is usually described under the names of Touraine, Solente and Golé in France, Switzerland and South America at the present time.

Several unusual and apparently distinct conditions in which clubbing of the fingers is a prominent symptom have become recognized. Pachydermoperiostosis and 'thyroid' acropachy [7] are two, and recently another type has been reported [12] in which clubbing is associated with a proliferative condition of the mucosa of the bladder with a mucous diarrhoea. Pachydermoperiostosis and the thyroid acropachy may not be entirely separate, since we found a low basal metabolic rate in our case. Dupont [4] has evidently also found a lowered basal metabolism in pachydermoperiostosis. However, the pachydermia of the upper eyelids in pachydermoperiostosis is not comparable with the tense oedema of the upper and lower lids and the progressive exophthalmos seen in 'thyroid' acropachy. The patient gave us no opportunity to test the effect of thyroid extract, but we gather that the basal metabolism may thereby be raised without improving the general condition.

When one notes the enlarged left ventricle and the warmth of the hypertrophic tissues in our patient, and the high pulse pressures with medial hypertrophy of the arteries which have been observed by others,[3] it seems that circulatory conditions analogous to those of Paget's disease may also be present in pachydermoperiostosis.

Our patient had slightly elongated extremities, although his stature was short. One cannot regard this as indicating a relationship to acromegaly, and it is moreover known [10] that pachydermoperiostosis is found in people who are somewhat long-limbed.

The occurrence of decided proliferative changes in the terminal phalanges is usually taken as a point against the diagnosis of pachydermoperiostosis, but it seems that this is not applicable to an advanced case such as ours.

SUMMARY

An advanced case is recorded in a South African Native of a syndrome known as pachydermoperiostosis, in which thick furrowed skin is found particularly on the forehead, upper eyelids and palms, associated with hyperostosis and osteophytosis of the limb bones and clubbing of fingers and toes. This condition is considered to be distinct from acromegaly and hypertrophic pulmonary osteo-arthropathy. The low basal metabolic rate in our case recalls the condition of clubbing of the fingers with periostosis which is known to occur after thyroidectomy (thyroid acropachy). With this disorder, however, a malignant exophthalmos is apt to be associated. Pachydermoperiostosis has not been found to be associated with any constant structural or functional disorders of the viscera.

although our case exhibited an unexplained left ventricular hypertrophy.

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NEW PREPARATIONS AND APPLIANCES

LUDOZAN TABLETS WITH BELLADONNA

Description. Ludozan tablets with belladonna—'Schering', contain hydrated sodium aluminium silicate with belladonna alkaloids. Designed for the treatment of more resistant cases of peptic ulcer. Ludozan tablets with belladonna contain 0.055 mg. alkaloids of belladonna per tablet. The addition of belladonna alkaloid is helpful in retarding the formation of hydrochloric acid by depression of the parasympathetic nerve endings. This vagus inhibition also results in the reduction of gastric motility and the relief of pylorospasm, factors of importance in the treatment of peptic ulcer.

Indications. The rapid relief afforded by Ludozan tablets in hyperacidity and peptic ulcer is due in large part to the prompt control of excess acidity. Ludozan tablets do not, however, abolish gastric acidity but only adjust the pH to within the physiological range. Therapy is, therefore, not attended by the danger of subsequent acid rebound.

Action. Ludozan tablets with belladonna afford mechanical protection of the ulcerated mucosa by coating the surface intimately with extremely finely divided particles of hydrated sodium aluminium silicate. These finely divided Ludozan particles have a powerful adsorptive action on excess pepsin and other irritants. Ludozan tablets with belladonna provide a true colloidal gel, at gastric pH which cannot be completely dissolved by hydrochloric acid and remains in the stomach to afford continuous protective effect. With Ludozan tablets with belladonna, administration is simple and convenient. An outstanding feature is the rapidity with which the tablets disintegrate in water.

Dosage. In peptic ulcer and as an antacid, one to two tablets, four or five times daily between meals and at bed time. In hyperacidity one to two tablets, three times daily after meals. As a protective gastric gel, one to two tablets half an hour before meals. The tablets may be chewed or swallowed (with water if required), or allowed to disintegrate in half a glass of water before taking.

How supplied. Packed in tubes of 10 tablets and bottles of 50 tablets. Manufactured in the Union of South Africa by Scherag (Pty.) Ltd., 127/129 Anderson Street, Johannesburg, for, and under the formula and technical supervision of, Schering Corporation, Bloomfield, N.J., U.S.A.

REVIEWS OF BOOKS

THE BENDER-GESTALT TEST

The Bender-Gestalt Test. By Gerald R. Pascal, Ph.D., and Barbara J. Suttell, M.S. (Pp. 274 + xiii, with graphs and illustrations. $6.50.) New York: Grune & Stratton, Inc. 1951.

Contents: Part I: Quantification. 1. Introduction. 2. Administration and Scoring. 3. Reliability. 4. Test Standardization. 5. Test Validity. Part II: Clinical Use of the Bender-Gestalt Test. 6. Introduction. 7. The Records of Children and Those with Cortical Deficit. 8. The Bender-Gestalt Test and Psychogenic Disorders. Appendix. Scoring Manual: 1. Introduction. 2. Definitions and Evaluations of Scoring Deviations. 3. Score Sheets. 4. Atlas of Scored Records. Index.

Since the publication of Dr. Bender's monograph *A Visual Motor Gestalt Test and its Clinical Use* in 1938, this test has been in widespread use. It has been employed to estimate maturation, intelligence, psychological disturbances and the effects of injury to the cortex, and to follow the effects of convulsive therapy. This is, however, the first published attempt to quantify the Bender-Gestalt test. This book gives also the first systematic presentation of the differences in performance between the records of psychiatric patients with psychogenic disorders and non-patients (i.e. normal controls).

Here we have an attempt to present a valid and feasible method of scoring this test so that its potentialities may be better utilized.

The authors found that, in the populations tested by them, where the subjects were of normal intelligence and free from brain damage, the greatest number of deviations was found in psychotic subjects, fewer in psychoneurotic subjects, and least in non-patients. They also make the statement "that the greater the damage to the cortex through convulsive therapy, amentia, lack of maturation, trauma, etc., the greater the deviations from the stimulus, and on our scoring system the higher the score on the B-G. test" (p. 9).

The findings here suggest to the authors that the deviations scored do not (for the normative population) measure drawing ability or I.Q. within average limits, but the test scores are correlated with whether or not an individual is the patient of a psychiatrist. The necessity for clinical judgment in addition to the use of these results is obvious.

This method of scoring is not feasible for children below six years of age, and even with older children the results are not as reliable as in the case of adults.

Each of 45 designs is reproduced and the deviations considered.

The book is extremely well produced and provides interesting reading. Those who accept the author's theory and interpretation will find it an essential addition to their library; and to the more sceptical one may say at the very least that it is a book well worth looking into.

It also suggests an interesting field of research in this country with regard to the use of the Bender-Gestalt test, particularly in the non-European population.

BRUCELLOSIS: ZOONOSES

Joint FAO: WHO Expert Panel on Brucellosis. Report on the First Session. World Health Organization: Technical Report Series No. 37. (Pp. 34. 2s. $0.25.) Geneva: World Health Organization, May 1951.

Joint WHO: FAO Expert Group on Zoonoses (Bovine Tuberculosis — Q Fever — Anthrax — Psittacosis — Hydatidosis). Report on the First Session. (Pp. 47. 2s. 3d. $0.30.) Geneva: World Health Organization, May 1951.

These two authoritative reports will be of great interest to medical practitioners. They comprise Reports on the first session devoted to the topics indicated. The zoonoses are obviously of veterinary public health importance and have an important bearing on the well-being and efficiency of man.

The Report dealing with Brucellosis not only has an important section on the standardization and interpretation of the sero-agglutination test, but also includes important information about the role of international agencies in brucellosis work.

CLINICAL ELECTROCARDIOGRAPHY

Basic Principles of Clinical Electrocardiography. By Hans H. Hecht, M.D. (Pp. 88 + ix. With 32 figures. 15s.) Illinois, U.S.A.: Charles C. Thomas. Oxford, England: Blackwell Scientific Publications.

Contents: 1. Introduction. 2. Unipolar Semi-Direct Leads. 3. Unipolar Limb Leads. 4. Bipolar Limb Leads. 5. Conclusions.

In this little monograph, publication No. 87 in the *American Lecture Series*, Hecht sets out to describe the basic principles by limiting his discussion as far as possible to 'practical considerations and factual evidence'. In spite of his intention to avoid discussion of membrane phenomena, he finds it necessary to consider the latter in the interpretation of the T wave.

Three basic patterns are described—the endocardial, the left ventricular epicardial, and the right ventricular epicardial. These patterns and their combinations determine the electrocardiogram. The interrelationship between the direct leads, the semi-direct leads, the unipolar limb leads, the standard bipolar limb leads and the vectorcardiographic curves is clearly set out.

To those who have difficulty in understanding modern electrocardiography this little book will be of great help. The text is lucid and will be easily followed with the possible exception of the section on the ventricular gradient.

The printing is good, and the figures are clear.

The book ends with a glossary for the learner, and a useful bibliography for those interested in further study.

HANDBOOK OF OPHTHALMOLOGY

A Handbook of Ophthalmology. By Humphrey Neame, F.R.C.S., and F. A. Williamson-Noble, F.R.C.S. (Pp. 338 + xi with 13 plates and 155 figures. Seventh Edition. 22s. 6d.) London: J. & A. Churchill Limited. 1951.

Contents: 1. Examination of the Eye and Its Surroundings. 2. Refraction and Accommodation. 3. Eyelids and Lacrymal Apparatus. 4. Injuries of the Eyeball. 5. The Conjunctiva. 6. Cornea and Sclerotic. 7. Iris, Ciliary Body and Choroid. 8. The Lens. 9. The Vitreous. 10. The Retina. 11. The Optic Nerve. 12. Glaucoma. 13. Extrinsic Muscles. 14. Orbit. 15. Operations. 16. Ophthalmological Signs and Symptoms in General Diseases. 17. Eye Diseases in the Tropics. 18. Therapeutics.

The welcome appearance of the seventh edition of this well-known textbook (within two years of the previous edition) is sufficient proof of its growing popularity among undergraduate students and general practitioners.

As in their previous editions, the authors have used the greatest detail in their description of the commoner ocular diseases and their treatment. All references to the rarities of ophthalmology have been omitted and they have confined themselves to brief notes upon the uncommon affections.

Numerous small changes have been made, notably in the section devoted to therapeutics. The authors prefer a strong iodine solution in the cauterization of dendritic ulcers to the pure liquid carbolic acid, as it is less likely to produce scarring. The increased dosages of Penicillin recommended are in conformity with present trends and the use of other antibiotics such as Aureomycin and Streptomycin are described. The treatment of ocular syphilis is given more space and the book has been brought up to date with present developments.

This reviewer has no hesitation in confidently recommending this handbook to undergraduates and general practitioners.

THE HEBREW MEDICAL JOURNAL

The Hebrew Medical Journal. Editorial Office: 983 Park Avenue, New York 28, N.Y., U.S.A.

The appearance of the Spring issue, volume 1, 1951, of *The Hebrew Medical Journal* (*Harofé Haivri*) inaugurates the 24th successful year of its publication under the editorship of Moses Einhorn, M.D. Written in Hebrew, with English summaries, the Journal is a contribution to the development of the Hebrew medical literature, and thus aids the newly established Hebrew University Medical School in Jerusalem.

In the medical section, among the articles of interest are: *Brain Tumors—Prognosis Based on Their Morphology* by Dr. Joseph H. Globus of Mt. Sinai Hospital, *The Modern Approach to the Management of the Failing Heart* by Dr. A. B. Rimmerman of Chicago and *Modern Concepts in the Diagnosis and Treatment of Marital Infertility* by Dr. Abner I. Weisman of New York City.

Under the heading of *Israel and Health*, Dr. Chaim Berlin discusses *Venereal Diseases in Israel*, and the subject *The Care of Deaf-Mutes in Israel* is presented by Dr. Mordecai Hexter.

In the section *Old Hebrew Medical Manuscripts*, Dr. Zussmann Muntner of Jerusalem presents a treatise on *Persian Medicine and Its Relation to Jewish and Other Medical Science—Commemorating the Millennium of Avicenna*, and Dr. Leon Nemoy of Yale University writes on *Medical Material in the Code of Karaite Law of Elijah Bashyatchi*.

UROLOGY: 1950

The 1950 Year Book of Urology (October, 1949—October, 1950). Edited by William Wallace Scott, M.D., Ph.D. (Pp. 416 with 87 illustrations. $5.00.) Chicago: The Year Book Publishers. 1950.

Contents: 1. Urology in the Decade 1940-50. 2. General Considerations. 3. The Kidney. 4. The Adrenals. 5. The Ureter. 6. The Bladder. 7. The Prostate. 8. The Genitalia.

The *Year Book* series on any specialized subject is to-day an essential, for in it are found summaries of the more important communications on the particular subject during the year. The range of journals is very wide. In this *Year Book* it is interesting to find that all the important urological communications from the *South African Medical Journal* are summarized.

This *Year Book* has a new Editor, Dr. W. W. Scott of Johns Hopkins. He has maintained the high standard set by his predecessors and in one respect has improved on them. The opening chapter, which gives a bird's eye view of the progress in urology during the last 10 years, is an excellent innovation and gives a balanced view of modern trends. The subject matter, the illustrations and the editorial comments are extremely good.

This series can be read with profit by all urologists and those who deal with conditions on the borderline of urology, such as adrenal tumours, perinephric abscess and testicular conditions.

NEUROLOGY, PSYCHIATRY AND NEUROSURGERY

The 1950 Year Book of Neurology, Psychiatry and Neurosurgery (November 1949—October 1950). Edited by R. P. Mackay, M.D., N. D. C. Lewis, M.D. and P. Bailey, M.D. (Pp. 627 with 121 figures. $5.00.) Chicago: The Year Book Publishers, Inc. 1951.

Contents: *Neurology*: 1. A Decade of Progress in Neurology (1940-50). 2. Anatomy and Physiology. 3. Pathology. 4. Trauma. 5. Infectious Diseases: Meningitis; Encephalitis; Poliomyelitis. 6. Degenerative Diseases. 7. Cerebrovascular Disorders. 8. Convulsive Disorders. 9. Neuropathies and Neuralgia. 10. Diagnostic and Therapeutic Methods.
Psychiatry: 11. Psychiatry during the Decade 1940-50. 12. General Topics. 13. Child Psychiatry. 14. Schizophrenia. Affective Disorders and Miscellaneous Reactions. 15. Organic Disorders and Toxic Reactions. 16. Therapy: Psychotherapy; Insulin Shock Therapy; Electric Shock; Psychosurgery; Miscellaneous Therapeutic Procedures.
Neurosurgery: 17. A Decade of Neurosurgery (1940-50). 18. Intracranial Tumors. 19. Epilepsy. 20. Leukotomy. 21. Motor Disorders. 22. Pain. 23. Suppuration. 24. Hemorrage. 5. Electroencephalography. 26. Cerebral Circulation. 27. Roentgenology. 28. Intraspinal Tumors. 29. Herniated Disks. 30. Malformations. 31. Peripheral Nerves. 32. Cranial Nerves. 33. Sympathetics. 34. Miscellaneous.

The contents of this publication are well worth reading, especially for the general practitioner who wishes to remain abreast of the newest developments in all fields of medicine and surgery. The introductory article of each section, *A Decade of Progress*, gives clearly and briefly what progress has been made and also, to an extent, where science has failed. The references of all articles extracted have been noted, thus enhancing the value of this book.

Of special interest are the extracts on lobotomy and leucotomy under the headings of anatomy, physiology and neurosurgery. In the psychiatric section, the extract on cytological changes in nerve cells in dementia praecox is of great interest.

With the present trends of medicine in the field of psychosomatics, the chapter on psychoneuroses and psychosomatics makes very interesting reading.

It is a book one might well wish to have on one's bookshelf.

YEAR BOOK OF ENDOCRINOLOGY: 1950

The 1950 Year Book of Endocrinology. (January 1950–January 1951.) Edited by Willard O. Thompson, M.D. (Pp. 499. With 138 illustrations. $5.00.) Chicago: The Year Book Publishers, Inc. 1951.

Contents: 1. Developments in Endocrinology 1940-50. 2. The Pituitary. 3. The Thyroid. 4. The Parathyroids. 5. The Adrenals. 6. The Testes. 7. The Ovaries. 8. Diabetes Mellitus. 9. Potassium Metabolism. 10. Miscellaneous. Index.

It is an interesting commentary on the development of experimental endocrinology that the *Year Book Series* has now inaugurated a volume devoted exclusively to this subject.

This is a most valuable contribution to contemporary experimental investigation because it has become manifestly impossible for the average practitioner either to subscribe to the great variety of Journals necessary or to find the time to read through them carefully if he is to keep abreast of current developments. The volume opens with an extremely concise and comprehensive review of developments in the field of endocrinology in the last decade.

As was perhaps to be expected the sections on the pituitary (including of course, ACTH) and the adrenal account for a very considerable portion of the book.

The material is extremely well selected and attention is drawn to the important experimental observation that ACTH and Cortisone may actually be contra-indicated in tuberculosis. Experimental work on mice indicates that the tuberculotic process is enhanced by these two potent hormones. This observation is also of interest in connexion with the multitude of diseases at present attributed to the adrenal cortex, which is regarded as essential in the development of resistance to infection. In these circumstances, it is difficult to see why there should be an exacerbation of tuberculosis as a result of cortisone treatment.

There can be little doubt that Endocrinology in the *Year Book* series has come to stay and will be regarded as essential in the library of the physiologist as well as the physician.

MALARIA IN EQUATORIAL AFRICA

Malaria in Equatorial Africa: Report on the WHO Malaria Conference in Equatorial Africa. World Health Organization: Technical Report Series No. 38. (Pp. 72. 3s. 6d. $0.45.) Geneva: World Health Organization. April 1951. Available also in a French edition.

The report on the Malaria Conference in Equatorial Africa is now available as No. 38 of the *World Health Organization: Technical Report Series.*

This Conference, held at Kampala, Uganda, under the joint auspices of WHO and the Commission for Technical Co-operation in Africa South of the Sahara, brought together experts on malaria from all parts of the world. The report on their discussions gives a comprehensive picture of the prevalence and effects of malaria on the continent of Africa and suggests methods for the control, and possible eradication of what an earlier conference termed an 'insidious' disease.

Among the subjects treated in this report are: (1) the general distribution and prevalence of malaria in Africa, and the morbidity and mortality which it causes; (2) the species of plasmodia and characteristics of their strains, *Plasmodium falciparum* being named as the parasite most widely and evenly distributed throughout Equatorial Africa; (3) the species and subspecies of the vectors, including their bionomics; (4) hyperendemic malaria and its relation to immunity; (5) the economic importance of malaria in Africa; (6) methods of malaria control; (7) therapeutics; (8) research on malaria in Africa, including a list of the malaria-research projects now in progress; (9) organization and training of malaria-control personnel; and (10) international planning for malaria-control in Africa. Annexures to the report give suggestions for uniform reporting of field research in malaria control and a list of the conventional symbols for the representation of anopheles of the Ethiopian region.

Among the conclusions of the conference that may well re-shape public health policy in Africa south of the Sahara, are two on which agreement has been achieved between two contrasting schools of thought: first, the advisability of carrying out malaria control even in areas where the adult population has developed tolerance against the disease; and, secondly the efficiency of residual-insecticide methods in controlling African malaria.

In addition to presenting valuable technical information, the report on the Malaria Conference in Equatorial Africa shows the importance of international co-operation in large-scale malaria-control programmes. For, as Sir John Hathorn Hall, Governor of Uganda, pointed out in his opening address: 'Where there are no natural boundaries preventing the spread of an insect-borne disease, its eradication from a continental area will clearly require the most co-ordinated form of human effort'.

NURSES' MEDICAL DICTIONARY

Baillière's Nurses Medical Dictionary. By M. Hitch, S.R.N. (Pp. 496 with 180 figures. 12th ed. 5s.) London: Baillière, Tindall & Cox. 1950.

Contents: 1. Foreword. 2. Preface to the Twelfth Edition. 3. Dictionary of Medical Terms. 4. Ordinary Materials used as Sutures; Surgical Instruments and their Care. 5. Anaesthetic Apparatus, including Apparatus for Gas-Air Analgesia and Oxygen Inhalation. 6. Technique for Ward Dressings. 7. Weights and Measures. 8. Thermometers. 9. Average Weights and Heights. 10. Abbreviations of Terms used in Prescriptions, etc. 11. Pharmacy and Poisons Act, 1933. 12. Common Drugs and Dosages. 13. Methods of giving Drugs, Alteration of Doses, etc. 14. Antibiotic Drugs. 15. Antiseptics and Disinfectants. 16. Urine Testing. 17. Food Values, Dietetics. 18. Poisoning. 19. Blood Transfusion. 20. First-Aid Treatment. 21. General details of preparation for X-ray Photography. 22. Degrees, Diplomas, etc. 23. Directory of Useful Addresses. 24. Splints, Bandages and Bandaging.

This book, in its small compact form, is a mine of information for nurses, for not only is it a sound dictionary containing all the terms which a nurse may need in the course of her studies and work, but it also has 21 appendices which take up one-third of the book space.

A glance at the list of contents will give an indication of the value of these appendices, and they are set out in sufficient detail to form in themselves a useful book.

Tabulation methods have been used where possible and while the dictionary section contains a number of diagrams explanatory of the text, some of the appendices are lavishly illustrated, especially those dealing with surgical instruments and anaesthetic apparatus.

This book is now in its twelfth edition and has been brought completely up to date.

PAEDIATRIC MEDICAL EMERGENCIES

Handbook of Pediatric Medical Emergencies. By A. G. DeSanctis, M.D. and C. Varga, M.D. (Pp. 284. With 51 illustrations. 42s. 6d.) St. Louis: The C. V. Mosby Company. 1951.

Contents: 1. Cardiovascular Emergencies. 2. Gastrointestinal Emergencies. 3. Genitourinary Emergencies. 4. Neurological Emergencies. 5. Respiratory Emergencies. 6. Drowning. 7. Poisoning. 8. Care of the Premature Infant. 9. Miscellaneous Emergencies. 10. Pediatric Procedures (illustrated). Agenda. Appendix.

The material in this handbook was prepared over a period of years to serve as a guide for members of the resident staff of the Bellevue Medical Centre, New York University. General revision was undertaken before the present wider publication. A most useful book has been produced, more particularly for those in charge of paediatric wards in hospitals.

To deal adequately with a medical emergency, the facilities of a modern hospital are usually essential. An attempt has been made to include within the scope of this book all those conditions in paediatric practice where urgent treatment might be required and the necessary full practical details are provided.

The methods described are based on the long personal experience of the authors and their collaborators, and have been kept thoroughly up to date. Dogmatic statement is unavoidable, and perhaps necessary, in a work of this nature; but the adopted methods are generally in keeping with accepted views.

A well-illustrated section on practical procedures is a valuable addition while references (almost exclusively American) to the paediatric literature are appended.

SURGERY FOR NURSES

Modern Surgery for Nurses. Edited by F. Wilson Harlow, M.B., B.S., F.R.C.S. With a Foreword by Sir Lancelot E. Barrington-Ward. (Pp. 799 + xxiv with 429 figures. 2nd ed. 25s.) London: William Heinemann Medical Books Limited. 1951.

Contents: Surgery: 1. Haemorrhage and Surgical Shock. 2. Saline and Glucose Infusions and Blood and Plasma Transfusions. 3. Fractures and Injuries to Bone. 4. Special Fractures. 5. Fractures of the Skull and Head Injuries. 6. Sprains and Dislocations. 7. Wounds and Superficial Injuries: Penicillin Therapy: Streptomycin. 8. Acute Inflammation and Infective Gangrene: Sulphonamide Treatment. 9. Chronic Inflammation: Tuberculosis, Syphilis and Actinomycosis. 10. Surgical Bacteriology. 11. Tumours and Cysts. 12. Ulcers, Sinuses and Fistulae: Skin-Grafting. 13. Burns and Scalds. 14. Diseases of the Veins and Arteries: Gangrene. 15. Peritonitis. 16. Appendicitis, and Diseases simulating Appendicitis. 17. The Oesophagus, Stomach and Duodenum. 18. The Gall-Bladder and Bile Ducts, Liver, Pancreas and Spleen. 19. Hernia and Intestinal Obstruction. 20. The Rectum and Anal Canal. 21. Urinary Cases: Special Instruments and Diagnostic Investigations. 22. The Kidneys and Ureters. 23. The Bladder and Prostate. 24. The Urethra and Male Genital Organs. 25. The Chest and Heart. 26. The Breast. 27. The Neck and Thyroid Gland. 28. The Face, Salivary Glands, Mouth, Tongue and Teeth. 29. The Throat and Nose. 30. The Ear and Head. 31. The Examination of Urine and other Diagnostic Tests in Common Use. 32. X-ray Diagnosis. 33. Preparation for Operation and Methods of Anaesthesia. 34. Surgical Technique. 35. Difficulties and Complications following Anaesthesia and Operation. 36. Later Post-Operative Complications and Complications during Convalescence. 37. The Care and Treatment of Special Cases. *Orthopaedics*: 38. Inflammatory Diseases of Bones and Joints. 39. Deformities, Congenital and Acquired. 40. Affections of the Spine. 41. Nerve Lesions. 42. Tumours and Non-Infective Diseases of Bone. *Gynaecology*: 43. Gynaecological Anatomy: Normal and Abnormal Menstruation. 44. Diseases and Displacements of the Uterus. 45. Anomalies and Complications of Pregnancy: Infections of the Upper Genital Tract. 46. Infections of the Lower Genital Tract and Diseases of the Vulva and Vagina. 47. Cysts and Tumours of the Ovaries, Sterility. Gynaecological Instruments, Pre- and Post-Operative Treatment of Gynaecological Cases. *Ophthalmic Surgery*: 48. Ophthalmic Surgery and Nursing. *Venereal Diseases*: 49. Syphilis, Gonorrhoea and other Venereal Diseases. *Anaesthetics*: 50. General Anaesthesia. 51. Intravenous Anaesthesia: Basal Narcosis and Rectal Anaesthesia. 52. Spinal Anaesthesia: Local, Regional, and Sacral Anaesthesia. *Appendix*: 53. Radiotherapy in the Treatment of Malignant Tumours: Surgical Electrical Instruments. 54. Useful Tables and Clinical Details. 55. The Sulphonamides and Antibiotics.

This book, which was first published in 1948, soon showed its worth as a combination of textbook and reference book for nurses, supplying a complete survey of modern surgery and surgical procedures. A year later it was reprinted with revisions, and this year a second edition has become necessary. It has now been thoroughly revised. Both additional material and a number of new illustrations have been added. The chapter on *X-Ray Diagnosis* has been re-written, while the chapters on *The Chest and Heart* and *Venereal Diseases* have been revised. The latest advances in antibiotic therapy form the subject of a new chapter. There is a great deal of other new material, among which will be found details of the reactions to blood transfusions, lumbar sympathectomy and vagotomy.

The book is well illustrated and contains a considerable amount of information; but when one realizes just how much knowledge is required of a nurse these days, one understands the need for a book such as this which covers the course so completely.

PASTEUR: THE LONE WOLF OF SCIENCE

Louis Pasteur—Free Lance of Science. By Rene J. Dubos. (Pp. 418. 21s.) Cape Town: Juta and Company Limited. London: Victor Gollancz Limited. 1951.

Contents: 1. The Wonderful Century. 2. The Legend of Pasteur. 3. Pasteur in Action. 4. From Crystals to Life. 5. The Domestication of Microbial Life. 6. Spontaneous Generation and the Role of Germs in the Economy of Nature. 7. The Biochemical Unity of Life. 8. The Diseases of Silkworms. 9. The Germ Theory of Disease. 10. Mechanisms of Contagion and Disease. 11. Medicine, Public Health and the Germ Theory. 12. Immunity and Vaccination. 13. Mechanisms of Discoveries. 14. Beyond Experimental Science. Events of Pasteur's Life Arranged in Chronological Order. Bibliography. Index.

This is one of the most readable, instructive and interesting accounts of Pasteur the man and Pasteur the scientist that has yet been penned. His remarkable ebullience, his robust intellect and his unquenchable thirst to satisfy an almost insatiable biological curiosity are outlined by Dr. Dubos in a masterly fashion.

The author's own writing pays an unconscious tribute to this great master in that it tells his story in the thorough way in which Pasteur himself might have done. There is none of the tendency (which Pasteur himself so vigorously castigated) to discuss a subject in 'florid and eloquent language instead of the factual statements of experimental science'; nor is there the temptation to engage in unfounded and unwarranted generalizations.

The contemporary scene probably no longer provides opportunities for such rugged individualists of scientific research as Pasteur. Although the irritation which men like Claude Bernard shared with Pasteur at their medical colleagues probably still finds expression, it is difficult to-day to realize what a lone battle Pasteur fought to establish the germ theory of disease. Dubos draws attention to the fact that the unassuming Darwin had as a disciple and exponent such a master of logic and invective as Huxley, whereas Pasteur had to play both these roles.

Readers will be interested in the story that in 1882 Pasteur was offered 100,000 francs by a Dutch financier for the exclusive right to the technique to manufacture anthrax vaccine for South Africa. Pasteur decided against the offer because he was impressed by the disastrous manner in which Liebieg had allowed his name to become associated with a meat extract.

The adequate and competent way in which the author sketches the environment in which Pasteur lived and moved is a tribute to his own thorough grasp of the social affairs which shaped Pasteur's society and his destiny.

The accurate presentation of the scientific material makes the book of abounding interest to the medical practitioner, but the presentation is a sufficiently lucid one to be fascinating and interesting for the lay reader as well.

MODERN TREATMENT: 1951

Modern Treatment Yearbook 1951. Edited by Sir Cecil Wakeley, K.B.E., C.B., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S. (Hon.). (Pp. 360 + viii. With 61 illustrations. 17s. 6d.) London: Published for The Medical Press by Baillière, Tindall and Cox, 1951.

Contents: 1. Cancer of the Stomach. 2. The Value of Corneal Grafts for Ophthalmic Lesions. 3. Streptomycin—Its Uses and Limitations in Treatment. 4. The Management of Cardiac Emergencies. 5. Some Medical Emergencies in Infancy and Childhood. 6. The Treatment of Flat Foot. 7. Cancer of the Uterus. 8. Modern Trends in the Treatment of Pulmonary Tuberculosis. 9. Treatment of Flail Foot. 10. Respiratory Emergencies. 11. The Principles and Practice of Psychological Therapy with Children. 12. Modern Surgical Treatment of Bronchiectasis in Children. 13. Early Diagnosis and Treatment of Congenital Dislocation of the Hips. 14. Acute Infection of the Middle Ear Tract. 15. Contracted Toes and Callosities. 16. The Place of the Chest Clinic in the Tuberculosis Service. 17. Hallus Valgus. 18. Modern Trends in Plastic Surgery. 19. The Place of Vagotomy in the Surgical Treatment of Chronic Peptic Ulcer. 20. Modern Treatment of Cancer of the Prostate. 21. Carcinoma of the Breast and its Treatment. 22. The Management of the Diabetic Child. 23. Modern Methods of Induction of Labour. 24. Medical Emergencies in Diabetes. 25. Differential Diagnosis in Diseases of the Chest. 26. Treatment of Cancer in the Oral Cavity. 27. Modern Trends in Occupational Therapy. 28. The Modern Treatment of Volkmann's Ischæmia. 29. Modern Treatment of Psoriasis. 30. Surgical Treatment of Sequelæ of Poliomyelitis. 31. The Management of Hypertension. 32. The Modern Treatment of Genital Prolapse. 33. Modern Treatment of the Anæmias. 34. Laryngeal Tuberculosis Associated with Pulmonary Tuberculosis: Its Incidence, Prognosis and Treatment. 35. The Diagnosis and Treatment of Cataract in Children. 36. The Out-Patient Treatment of Fractures. 37. Modern Trends in the Treatment of Skin Diseases. 38. Insomnia in Children. 39. The Diagnosis and Treatment of Perforated Peptic Ulcer.

Sir Cecil Wakeley's claim that 'almost the whole field of medicine and surgery has been covered by the articles contained in this book' is a very just one. It remains platitudinous but never-the-less true, that diagnosis is an essential prerequisite for adequate and effective treatment. From this point of view this volume performs a double function in that it is of diagnostic as well as therapeutic assistance to the busy general practitioner.

Clearly printed and attractively illustrated, this volume will make a strong appeal to the conscientious general practitioner.

CORRESPONDENCE

THE 'TICKEY-A-WEEK DOCTOR'

To the Editor: In recent months much thought has been given to the sad decline in the status of the General Practitioner. The G.P. viewed the problem from every angle, but never introspectively; forgetting that 'The fault, dear Brutus, lies not with our stars, but in ourselves that we are underlings'.

The fact that there is so large a proportion of specialists in South Africa seemed to him to have some bearing on the problem.

Actually, this decline in status is world-wide, it existed in Great Britain in the N.H.I. days, in centres where very few specialists had established their practices. Cities comparable with Cape Town in size and population had two surgeons, one physician, one E.N.T. surgeon, a gynaecologist and perhaps a paediatrician. The only dissimilar factor which may have some bearing on this matter, in conditions as they exist in South Africa, is that here the profession agreed to the establishment of a specialist register for certain doctors, compared with whom doctors on the general register were willy-nilly considered to be of lesser status.

There can be no respect in the absence of self-respect. In the past the doctor was held in high esteem by his patient and though his skill and knowledge could not compare with that of his modern counterpart, the patient who paid a fee for his services was making some sacrifice and never doubted that he got value for his money.

In actual purchasing power the fee was very much higher than it is to-day and the patient set a value on the services commensurate with the fee which he was called upon to pay; no man will willingly admit that he pays more for any commodity than it is worth. To-day the G.P. is held in esteem by just those patients who directly, or through Medical Aid Groups, pay him for the services he renders. The one factor which is almost exclusively responsible for the decline in status is the establishment universally of Medical and Sick Benefit Societies from National Health Insurance and Sick Funds down to small Benefit Societies with memberships of less than 50 members.

Is there any other calling or profession whose members will bind themselves for a tickey a week, sometimes less, to be at the beck and call of a fellow-being at any time of day or night? What respect can a patient possibly have for a doctor who is prepared to bind himself in this fashion for him? What self-respect can the poor doctor retain?

The remedy lies in the fostering of the development of Medical Aid Societies which allow free choice of doctor and offer a fee per item of service rendered.

I fear I have wakened my Muse and append the lament of one such 'Arme Ou':

O! rus my siel, jou sorge oor,
Slaap veilig deur die nag
Van nou af kan ons tafel dek
En kleed en kos verwag.

Met talle van ons medemens
Is ons tans vas gebind
Om alle dienste te verskaf
Aan vrou en man en kind.

Ons staan gereed beid' nag en dag
Deur hitte, wind en reën
Aan elke roep gehoor te gee
En hulp daaraan te leen.

Meen nie ons doen dit sonder loon,
Met engele vergelyk
Elkeen sit weekliks 'tiekie' in
En daarvan word ons ryk.

Wat kos dit, O! my dierbaar siel?
Vir vrede so genoeg.
Van ons word dit beslis verwag
Dat ons iets by moet voeg.

Ons eer, ons rustyd, selfrespek
Lê alles in die skaal
Om man en vrou tevreed' te stel
Wat 'trippens' elk betaal.

Kan selfrespek, nog eer, bestaan?
Met leë maere beurs
Teen dringend nood en hongersknaag
Waar geld as koning heers.

O! swyg my siel, bekommer nie;
Ek hoor daar word gesê
Vol loon is aan ons toegedien
As ons ter ruste lê.

J. H. L. Shapiro.

Railway Medical Office,
Dock's Surgery,
Cape Town.
23 August 1951.

DISTRICT SURGEONS AND DRIVERS UNDER THE INFLUENCE OF ALCOHOL

To the Editor: It is high time something was done about this ridiculous business of asking District Surgeons to certify whether certain car drivers are under the influence of alcohol and incapable of driving a car at the time of accidents. No particular medical knowledge is required for this purpose; whereas the opinion of any sensible layman, especially an experienced policeman, should be just as reliable, in fact more so, as the district surgeon usually sees his man long after the accident when he has had plenty of time to sober up. It is surprising how soon they sober up after an accident.

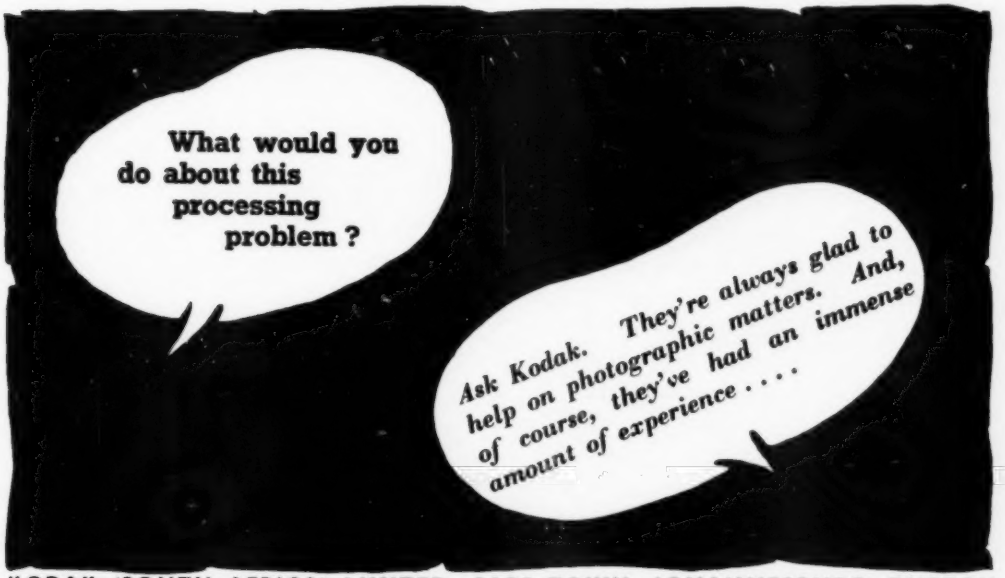
A policeman, finding an accident, should stay on the spot to collect evidence and not be obliged to rush away with the culprit to find a district surgeon that could examine the case before he has time to sober. Is the district surgeon standing on his doorstep waiting? I think we all know that a medico is often the most difficult man to find. Hence what a search and what a waste of time!

Thus, is there any one reason why a district surgeon should be called upon to do such a thankless and most odious job? It is against his nature to condemn. He likes to be the friend of everybody, so that even the worst criminal can trust a medical man as his professional friend and not his enemy. Often, especially in a small country town or dorp, the district surgeon finds himself called upon to pass judgment on one of his best clients, or a member of a family containing such clients or even, perhaps, his true and personal friends. I know of a case where a district surgeon, after giving such evidence, had to walk out of court, straight to the timid and nervous wife of the accused to help her give birth to a child! Is that right? Can such a medical man's evidence be regarded as unbiassed? We must remember that not all medicos are angels and, I daresay, even angels might find difficulty in deciding between duty to the State and the public and duty towards protecting the health and nerves of the poor, sickly, frail and anxious wife, mother or daughter, especially seeing that they are accepting money for the latter duty.

Of course there is one snag. Certain pathological conditions might, on rare occasions, give rise to symptoms resembling alcoholic intoxication, but surely that could easily be remedied by the simple expedient of asking the district surgeon, before locking up the culprit, to certify that the patient is not suffering from any disease, etc., simulating intoxication.

L. Lappin.

Villiersdorp,
Cape Province.
8 September 1951.



The Medical Association of South Africa
Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP AFDELING

JOHANNESBURG

Medical House, 5 Esselen Street. Telephones 44-9134-5, 44-0817
Mediese Huis, Esselenstraat 5. Telefone 44-9134-5, 44-0817

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(Pr S30) Johannesburg Partnership practice plus Solus practice. Mainly non-European. Present income £3,600 p.a. Premium for quick sale £1,250.

(Pr S31) O.V.S.-praktyk. Goeie geleentheid vir algemene geneesheer met aanleg vir snywerk. Alle fasiliteite. Medisyne word aangemaak. Moet tweetalig wees. Jaarliks inkomste £2,400. Eienaar gaan verder studeer. Premie vir klandiesiewaarde, instrumente en voorrade, £1,500. Een maand introduksie sal gegee word.

MEDICAL EQUIPMENT

(I 017) Leitz microscope £45.

(I 018) What offers? Complete set *British Encyclopaedia of Medical Practice* plus all editions of *Medical Progress* to 1950. Condition as new.

(I 019) Zeiss microscope. Condition as new. £55.

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* * * *

KAAPSTAD : CAPE TOWN

Posbus 643, Telefoon 2-6177 : P.O. Box 643, Telephone 2-6177

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(350) Eastern Cape hospital town. Total gross receipts for preceding 13 months £3,700. One appointment. Premium of £2,000 includes drugs, surgery furniture, fittings, etc. House for sale at £3,000. Large bond available. £700 rebate if appointment not transferred. Practice offers great scope for practitioner with surgical ability.

(644) Durban Central. Mainly Indian and Native cash practice. Average annual gross income £1,235. Premium of £500 required for goodwill, inclusive of furniture and fittings and drugs. Terms may be arranged.

(805) Transkei Native and D.S. practice. Near large town. House and surgery for sale, purchaser preferably bilingual.

(821) Eastern Province hospital town. Gross cash receipts £2,200. Premium required £850 which includes drugs, furniture and instruments valued at £350. Mainly non-European at present but with definite scope for future.

VENNOOTSKAP VERLANG : PARTNERSHIP REQUIRED

(811) Partnership share in Cape or Natal in predominantly English-speaking practice with min. net. income £2,500 p.a.

ASSISTENTE/PLAASVERVANGERS VERLANG ASSISTANTS/LOCUMS REQUIRED

(689) Zululand Mission Hospital with 60 beds. Locum tenens, man or woman, for 12 to 18 months. Travelling expenses paid and furnished house available. Salary to be discussed.

(809) Gentile assistant for Transkei general practice with D.S. appointment. Single man preferred. Excellent opportunity to gain sound experience. Salary to be arranged.

(780) Transkei hospital town. For month December. £2 2s. p.d. plus hotel expenses. Car provided. Practice with D.S. appointment.

MEDICAL EQUIPMENT FOR SALE

(772) Strand, C.P. Couch, instrument and dressing tables, cupboards and waiting-room furniture, at approx. £100. Instruments at £100.

(758) Electrocardiograph. Sanborne Cardiette. Weight 24 lb. Perfect working condition. Used by Cape Town specialist physician. £160 or nearest offer.

(674) Complete up-to-date set of the *British Encyclopaedia of Medical Practice*. Any reasonable offer.

Johannesburg Hospital and the University of the Witwatersrand

VACANCIES FOR FULL-TIME OBSTETRICIAN AND GYNAECOLOGIST, (SENIOR) AND POSSIBLE CONSEQUENTIAL VACANCIES

A vacancy will shortly exist in the department of obstetrics and gynaecology for a full-time obstetrician and gynaecologist (senior) who will be the senior professional assistant to the Professor of obstetrics and gynaecology.

Applications are invited for this post and/or for any consequential vacancies which may arise as full-time obstetrician and gynaecologist or full-time obstetrician and gynaecologist (assistant) in the event of a member of the present medical staff being promoted within the department as a result of the consideration of these applications.

The salary attached to the grade of full-time obstetrician and gynaecologist (senior) is £2,000 per annum, to that of full-time obstetrician and gynaecologist is £1,800 per annum, and to that of full-time obstetrician and gynaecologist (assistant) is £1,200 × 50—£1,500 per annum, plus in each cost-of-living allowance at the current rate in force.

Applicants should preferably submit their applications on the official form T.A. 633 obtainable on application to the Provincial Secretary, Hospital Services Branch, P.O. Box 383, Pretoria or to the Medical Superintendent, Johannesburg Hospital.

If unable to do so, they must at least state full name, address, date and place of birth, marital state, language qualifications, academic experience and the earliest date on which they can assume duty. Copies only of testimonials should be sent.

Applications must be submitted in duplicate and be addressed to the Medical Superintendent, Johannesburg Hospital, Smit Street, Johannesburg, and must reach his office not later than 4 p.m. on 22 October 1951. (31359)

Cecil John Adams Memorial Trust

TRAVELLING FELLOWSHIPS

Mr. A. E. Adams has established a Trust for the endowment of Travelling Fellowships in memory of his son Cecil John Adams who lost his life in the recent war.

A Fellowship is of the annual value of about £500, and shall be tenable for one complete year of twelve months, but may be extended for a further period at the discretion of the Selection Committee. Four Fellowships are available.

Candidates must be South African graduates in medicine or medical science, they must have shown promise of being likely to profit from further study, research and experience in other countries and must have been resident in South Africa for at least three years immediately prior to the date of application for a fellowship.

Application (in triplicate) must be made on a prescribed form obtainable from the undersigned, and must reach the Trustee by 31 October 1951.

J. J. le Roux
Trustee

The South African Association
6 Church Square
Cape Town

Vulco Chemical Company Limited

PART-TIME MEDICAL OFFICER

Applications are invited for the position of part-time medical officer to the Vulco Chemical Co. Limited. For further particulars apply to the Secretary, P.O. Box 3754, Johannesburg.

Resident Medical Officer

Applications are invited for the above post by colliery in the Witbank area. House and surgery will be provided. Apply giving full particulars to 'A. I. L.', P.O. Box 643, Cape Town.

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

VACANCY—JUNIOR RESIDENT MEDICAL OFFICER (INTERN)

SIR HENRY ELLIOT HOSPITAL, UMTATA

Applications are invited from suitably qualified persons for the above-mentioned post. The salary applicable thereto is £240 per annum plus the cost-of-living allowance at Civil Service rates and free board, quarters and laundering.

The appointment will be on contract for a period of 12 months as from 1 January 1952 and may be extended by mutual agreement.

The appointment will be made in terms of and be subject to the Hospital Board Service Ordinance No. 19 of 1941 and the Regulations framed thereunder.

Applications on forms Staff 23 (in duplicate), which are obtainable from all Hospital Offices in the Cape Province, must be addressed to the Medical Superintendent, Sir Henry Elliot Hospital, Umtata, so as to reach him not later than noon on Friday, 30 November 1951.

L. B. Larter
Acting Branch Representative
(O. 1138)

P.O. Box 202
Umtata
27 September 1951

City of Johannesburg

VACANCIES

Applications are invited for the following positions in the City Health Department:—

(i) Senior dental officer—salary £1,104 per annum fixed.
(ii) Dental officers—salary £864 per annum fixed.

In addition to the basic salary a variable cost-of-living allowance is paid, at present £22 5s. 10d. per month.

The successful applicants will be required to undergo a medical examination and become members of the Council's Pension Fund.

Applicants must be dental surgeons registered to practice in South Africa. Further qualifications or experience in Public Dentistry will be a recommendation. Details of conditions of service will be supplied on application to the Medical Officer of Health, P.O. Box 1477, Johannesburg.

Applications in the candidates' own handwriting on special forms obtainable from the Central Staff Office, Room 223, Municipal Offices, must be placed in the box in Room 223, Municipal Offices, not later than 4 p.m. on 31 October 1951.

Brian Porter
Town Clerk
388
(A. 1865)

Transvaal Provincial Administration

VACANCIES: TRANSVAAL PUBLIC HOSPITALS

Applications are invited from suitably qualified candidates for the undermentioned posts at Public Hospitals in the Transvaal.

Applications should be addressed to the Medical Superintendents of the Hospitals concerned and should contain full particulars as to the age, professional, academic and language qualifications, experience and conjugal status of the applicant and should further indicate the earliest date upon which duties can be assumed. Copies, only of recent testimonials to be attached.

Pretoria:—Junior physician (1): £1,200 × 50—£1,500. Must be suitably qualified by experience and training. Plus cost-of-living allowance at current rates.

Ventersdorp:—Part-time general practitioner (1): £340 per annum. Two sessions per week.

Closing date of applications: 22 October 1951.

Application forms are obtainable from the Provincial Secretary, Hospital Services Department, P.O. Box 383, Pretoria. (31354)

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